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FILE COVERS 1907 - 11 Jul 2004  VOL 141 ISS 3
FILE LAST UPDATED: 9 Jul 2004  (20040709/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


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=> d stat que
L1            21 SEA FILE=REGISTRY ABB=ON  PLU=ON  FASCILIN? OR STABILIN?
L2           379 SEA FILE=REGISTRY ABB=ON  PLU=ON  CD44 OR CD(L)44
L3           418 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L1 OR ?FASCILIN? OR ?STABLILIN
                 ?
L4          4329 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L2 OR CD44 OR CD(W)44
L5         40848 SEA FILE=HCAPLUS ABB=ON  PLU=ON  FELL
L7             3 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3 AND (L4 OR L5)


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=> d ibib abs hitrn l7 1-3

L7   ANSWER 1 OF 3  HCAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         2004:80331  HCAPLUS
DOCUMENT NUMBER:          140:140710
TITLE:                    cDNAs encoding human NOVX proteins and their
                          diagnostic and therapeutic use
INVENTOR(S):              Shimkets, Richard A.; Patturajan, Meera; Vernet,
                          Corine A. M.; Casman, Stacie J.; Malyankar, Uriel M.;
                          Shenoy, Suresh G.; Spytek, Kimberly A.; Gangolli, Esha
                          A.; Miller, Charles E.; Boldog, Ferenc L.; Li, Li;
                          Taupier, Raymond J.; Kekuda, Ramesh; Smithson,
                          Glennda; Zerhusen, Bryan D.; Liu, Xiaohong; Colman,
                          Steven D.; Tchernev, Velizar T.; Si, Jingsheng;
                          Edinger, Shlomit R.; Stone, David J.; Sciore, Paul;
                          Millet, Isabelle; Rothenberg, Mark E.
PATENT ASSIGNEE(S):       USA
SOURCE:                   U.S. Pat. Appl. Publ., 240 pp., Cont.-in-part of U.S.
                          Ser. No. 28,248.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
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FAMILY ACC. NUM. COUNT: 140
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004018970	A1	20040129	US 2002-107782	20020327
US 2003235882	A1	20031225	US 2001-28248	20011219
US 2003203363	A1	20031030	US 2002-94466	20020307
EP 1427749	A2	20040616	EP 2002-713788	20020308

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.:

US 2000-256619P	P	20001219
US 2001-262959P	P	20010119
US 2001-272408P	P	20010228
US 2001-279344P	P	20010328
US 2001-285189P	P	20010420
US 2001-308039P	P	20010726
US 2001-311266P	P	20010809
US 2001-28248	A2	20011219
US 2001-274101P	P	20010308
US 2001-274194P	P	20010308
US 2001-274281P	P	20010308
US 2001-274322P	P	20010308
US 2001-274849P	P	20010309
US 2001-275235P	P	20010312
US 2001-275578P	P	20010313
US 2001-275579P	P	20010313
US 2001-275601P	P	20010313
US 2001-276000P	P	20010314
US 2001-276776P	P	20010316
US 2001-276994P	P	20010319
US 2001-277239P	P	20010320
US 2001-277321P	P	20010320
US 2001-277327P	P	20010320
US 2001-277338P	P	20010320
US 2001-277791P	P	20010321
US 2001-277833P	P	20010322
US 2001-278152P	P	20010323
US 2001-278894P	P	20010326
US 2001-278999P	P	20010327
US 2001-279036P	P	20010327
US 2001-279995P	P	20010330
US 2001-280233P	P	20010330
US 2001-280802P	P	20010402
US 2001-280822P	P	20010402
US 2001-280900P	P	20010402
US 2001-281194P	P	20010404
US 2001-283675P	P	20010413
US 2001-287424P	P	20010430
US 2001-288066P	P	20010502
US 2001-288148P	P	20010502
US 2001-288342P	P	20010502
US 2001-288528P	P	20010503
US 2001-291190P	P	20010515
US 2001-291099P	P	20010516
US 2001-291240P	P	20010516
US 2001-294485P	P	20010530
US 2001-294821P	P	20010531
US 2001-294889P	P	20010531
US 2001-294899P	P	20010531
US 2001-335302P	P	20011031
US 2001-338375P	P	20011204

AB The present invention provides cDNAs encoding human NOVX proteins and their diagnostic and therapeutic use.

IT **651797-66-9**, Protein (human stabilin-like) **651797-84-1**, Protein (human stabilin-like) **651797-86-3**, Protein (human **CD44** antigen-like)
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; cDNAs encoding human NOVX proteins and their diagnostic and therapeutic use)

IT **651797-65-8 651797-83-0 651797-85-2**
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; cDNAs encoding human NOVX proteins and their diagnostic and therapeutic use)

L7 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:696159 HCAPLUS
DOCUMENT NUMBER: 137:246071
TITLE: Gene expression profiles relating to normal and osteoarthritic cartilage
INVENTOR(S): Liew, Choong-Chin; Marshall, Wayne E.; Zhang, Hongwei
PATENT ASSIGNEE(S): Chondrogene Inc., Can.
SOURCE: PCT Int. Appl., 777 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 16
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002070737	A2	20020912	WO 2002-CA247	20020228
WO 2002070737	C1	20021031		
WO 2002070737	C2	20031002		
WO 2002070737	A3	20040129		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1404868	A2	20040407	EP 2002-703416	20020228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.:
US 2001-271955P P 20010228
US 2001-275017P P 20010312
US 2001-305340P P 20010713
WO 2002-CA247 W 20020228

AB The invention provides gene expression profiles comprising one or more polynucleotide sequences that are expressed in chondrocytes from any of the following developmental and disease stages: fetus, normal adult, mild osteoarthritis, moderate osteoarthritis, marked osteoarthritis, and severe osteoarthritis. Complementary DNA libraries were constructed from human fetal, normal, mild osteoarthritic and severe osteoarthritic cartilage samples (13,398, 17,151, 12,651, and 14,222 expressed sequence tags (ESTs), resp.). The known and novel clones derived from these libraries were then used to construct human chondrocyte-specific microarrays to generate differential gene expression profiles useful as a diagnostic

tools for detection of osteoarthritis. A total of 5807 expressed gene sequences are provided and matched to known gene sequences, other ESTs, or mitochondrial, ribosomal, vector, and cDNA/hypothetical protein sequences in the public databases. Arrays of the invention are useful as a gold standard for osteoarthritis diagnosis and for use to identify and monitor therapeutic efficacy of new drug targets.

IT **249596-99-4 249767-19-9 391528-58-8**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; gene expression profiles relating to normal and osteoarthritic cartilage)

L7 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:575239 HCAPLUS
DOCUMENT NUMBER: 137:136135
TITLE: Human cDNA sequences and their encoded proteins and diagnostic and therapeutic uses
INVENTOR(S): Shimkets, Richard A.; Patturajan, Meera; Vernet, Corine A. M.; Casman, Stacie J.; Malyankar, Uriel; Shenoy, Suresh; Spytek, Kimberly A.; Gangolli, Esha; Miller, Charles; Boldog, Ferenc; Li, Li; Taupier, Raymond J., Jr.; Kekuda, Ramesh; Smithson, Glennda; Zerhusen, Bryan D.; Liu, Xiaohong; Colman, Steven D.; Tchernev, Velizar; Si, Jingsheng; Edinger, Schlomit; Stone, David; Sciore, Paul; Millet, Isabelle; Rothenberg, Mark
PATENT ASSIGNEE(S): Curagen Corporation, USA
SOURCE: PCT Int. Appl., 363 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 140
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002059315	A2	20020801	WO 2001-US50076	20011219
WO 2002059315	A3	20031009		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 2000-256619P P 20001219
US 2001-262959P P 20010119
US 2001-272408P P 20010228
US 2001-285189P P 20010420
US 2001-308039P P 20010726
US 2001-311266P P 20010809

AB Disclosed herein are 20 cDNA sequences that encode novel human polypeptides that are members of the following protein families: stabilin, **CD44**-like precursor/**fascilin** domain, polydom, transmembrane IIIb protein, serine proteinase, Wnt-7a protein, apical endosomal glycoprotein, ADAM13, leucine-rich F box-containing protein, steroid-binding protein, steroid dehydrogenase, myosin heavy chain, and pancreatitis-associated protein. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivs., variants, mutants, or

fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

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=> select hit rn l7 1-3
E1 THROUGH E9 ASSIGNED

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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 9 JUL 2004 HIGHEST RN 707542-72-1
DICTIONARY FILE UPDATES: 9 JUL 2004 HIGHEST RN 707542-72-1

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 6, 2004

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Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. For more information enter HELP PROP at an arrow prompt in the file or refer to the file summary sheet on the web at:
http://www.cas.org/ONLINE/DBSS/registryss.html

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(FILE 'HCAPLUS' ENTERED AT 11:25:32 ON 11 JUL 2004)

FILE 'HCAPLUS' ENTERED AT 11:27:39 ON 11 JUL 2004

FILE 'HCAPLUS' ENTERED AT 11:28:38 ON 11 JUL 2004
SELECT HIT RN L7 1-3

FILE 'REGISTRY' ENTERED AT 11:29:01 ON 11 JUL 2004
L8 9 S E1-E9

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=> => d ide can l8 1-9

L8 ANSWER 1 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **651797-86-3** REGISTRY
CN Protein (human CD44 antigen-like) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 6: PN: US20040018970 SEQID: 211 claimed protein
CN DNA (human protein NOV1c cDNA plus flanks)

FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties); USES (Uses)

RELATED SEQUENCES AVAILABLE WITH SEQLINK

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 140:140710

L8 ANSWER 2 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **651797-85-2** REGISTRY
CN DNA (human CD44 antigen-like protein cDNA plus flanks) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 5: PN: US20040018970 SEQID: 210 claimed DNA
CN DNA (human protein NOV1c cDNA plus flanks)
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties); USES (Uses)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 140:140710

L8 ANSWER 3 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **651797-84-1** REGISTRY
CN Protein (human stabilin-like) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 4: PN: US20040018970 SEQID: 4 claimed protein
CN Protein NOV1b (human)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties); USES (Uses)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 140:140710

L8 ANSWER 4 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **651797-83-0** REGISTRY
CN DNA (human stabilin-like protein cDNA plus flanks) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 3: PN: US20040018970 SEQID: 3 claimed DNA
CN DNA (human protein NOV1b cDNA plus flanks)
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties); USES (Uses)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 140:140710

L8 ANSWER 5 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **651797-66-9** REGISTRY
CN Protein (human stabilin-like) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2: PN: US20040018970 SEQID: 2 claimed protein
CN Protein NOV1a (human)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties); USES (Uses)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
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1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 140:140710

L8 ANSWER 6 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **651797-65-8** REGISTRY
CN DNA (human stabilin-like protein cDNA plus 3'-flank) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: US20040018970 SEQID: 1 claimed DNA
CN DNA (human protein NOV1a cDNA plus 3'-flank)
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties); USES (Uses)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 140:140710

L8 ANSWER 7 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **391528-58-8** REGISTRY
CN DNA (human gene CD44 cell adhesion molecule cDNA plus flanks) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1170: PN: WO03038130 FIGURE: 3 claimed DNA
CN 155: PN: WO03083140 TABLE: 5 unclaimed DNA
CN 2248: PN: WO02070737 FIGURE: 6 unclaimed DNA
CN 2275: PN: WO03091391 TABLE: 20 unclaimed DNA
CN 228: PN: WO03081201 TABLE: 1 claimed DNA
CN 2452: PN: WO2004038376 TABLE: 5 unclaimed DNA
CN 374: PN: WO2004046386 TABLE: 5 unclaimed DNA
CN 4107: PN: WO2004037996 TABLE: 3 claimed DNA
CN 523: PN: WO2004024892 FIGURE: 7 unclaimed DNA
CN 55: PN: WO03100029 PAGE: 121 unclaimed DNA
CN DNA (human CD44 gene cDNA plus flanks)
CN DNA (human gene CD44 cDNA)
CN GenBank M59040
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: BIOSIS, CA, CAPLUS, GENBANK, TOXCENTER, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: ANST (Analytical study); BIOL (Biological study); PRP (Properties); USES (Uses)

RELATED SEQUENCES AVAILABLE WITH SEQLINK

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
14 REFERENCES IN FILE CA (1907 TO DATE)
14 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 141:21838

REFERENCE 2: 140:402911

REFERENCE 3: 140:373461

REFERENCE 4: 140:285716

REFERENCE 5: 140:26912

REFERENCE 6: 140:3792

REFERENCE 7: 139:302993

REFERENCE 8: 139:302953

REFERENCE 9: 139:291131

REFERENCE 10: 138:380506

L8 ANSWER 8 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **249767-19-9** REGISTRY

CN DNA (human CD44 (antigen) cDNA plus flanks) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1401: PN: WO02059377 TABLE: 4 claimed DNA
CN 1487: PN: US20040009479 TABLE: 3A unclaimed DNA
CN 1614: PN: WO02070737 FIGURE: 6 unclaimed DNA
CN 25: PN: US20040009479 TABLE: 8 unclaimed DNA
CN 375: PN: WO2004046386 TABLE: 5 unclaimed DNA
CN 4648: PN: US20040009481 TABLE: 1 claimed DNA
CN DNA (human gene CD44 cDNA plus flanks)
CN DNA (human gene CD44 cDNA)
CN GenBank AJ251595
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: CA, CAPLUS, GENBANK, TOXCENTER, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties)
RLD.P Roles for non-specific derivatives from patents: ANST (Analytical study); BIOL (Biological study); PRP (Properties); USES (Uses)

RELATED SEQUENCES AVAILABLE WITH SEQLINK

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
9 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
9 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 141:21838

REFERENCE 2: 140:234408

REFERENCE 3: 140:234407

REFERENCE 4: 140:194431

REFERENCE 5: 138:282467

REFERENCE 6: 137:246071

REFERENCE 7: 137:227709

REFERENCE 8: 137:152024

REFERENCE 9: 137:1484

L8 ANSWER 9 OF 9 REGISTRY COPYRIGHT 2004 ACS on STN
RN **249596-99-4** REGISTRY
CN DNA (human clone D87433 stabilin 1 cDNA plus flanks) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1627: PN: WO02070737 FIGURE: 6 unclaimed DNA
CN DNA (human clone D87433 gene stab1 stabilin-1 cDNA plus flanks)
CN DNA (human gene stab1 cDNA)
CN GenBank AJ275213
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: CA, CAPLUS, GENBANK, TOXCENTER, USPATFULL
DT.CA CAplus document type: Journal; Patent

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RL.P   Roles from patents:  BIOL (Biological study); PRP (Properties)
RL.NP  Roles from non-patents:  BIOL (Biological study); PRP (Properties)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
               3 REFERENCES IN FILE CA (1907 TO DATE)
               3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE   1:  139:111648

REFERENCE   2:  137:246071

REFERENCE   3:  137:74944


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FILE 'HCAPLUS' ENTERED AT 11:39:14 ON 11 JUL 2004
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FILE COVERS 1907 - 11 Jul 2004  VOL 141 ISS 3
FILE LAST UPDATED: 9 Jul 2004  (20040709/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


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=> d stat que
L1           21 SEA FILE=REGISTRY ABB=ON  PLU=ON  FASCILIN? OR STABILIN?
L2          379 SEA FILE=REGISTRY ABB=ON  PLU=ON  CD44 OR CD(L)44
L3          418 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L1 OR ?FASCILIN? OR ?STABLILIN
                ?
L4         4329 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L2 OR CD44 OR CD(W)44
L5        40848 SEA FILE=HCAPLUS ABB=ON  PLU=ON  FELL
L7            3 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L3 AND (L4 OR L5)
L10         981 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L3 OR L4 OR L5)(L)(?ENCOD?
                OR CODE? OR CODING OR HOMOLOG?)
L12          42 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L3 OR L4 OR L5)(4W)LIKE
L13           6 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L10 AND L12
L14           4 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L13 NOT L7
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=> d ibib abs hitrn l14 1-4

L14 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:537916 HCAPLUS
DOCUMENT NUMBER: 131:154496
TITLE: Protein and DNA sequences **encoding** a human **CD44-like** protein
INVENTOR(S): Ni, Jian; Gentz, Reiner L.; Dillon, Patrick J.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: U.S., 38 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5942417	A	19990824	US 1997-892880	19970715
US 2003105058	A1	20030605	US 2002-291634	20021112

PRIORITY APPLN. INFO.: US 1996-21762P P 19960715
US 1997-892880 A1 19970715
US 1999-288230 A1 19990408

AB The invention provides protein and DNA sequences of a novel **CD44-like** protein, which is about 24% identical and about 46% similar to rat CD44. CD44 is known to act as a receptor for hyaluronan, and the protein of the present invention is able to bind hyaluronan as well. The invention further relates to screening methods for identifying agonists and antagonists capable of enhancing or inhibiting **CD44-like** protein-mediated signaling, and therapeutic methods for treating diseases associated with said signaling.

IT **203673-50-1 203673-51-2**, **CD44** (antigen) (human clone HUVDE75) **203673-52-3 203743-82-2 237078-01-2**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(amino acid sequence; protein and DNA sequences **encoding** a human **CD44-like** protein)

IT **203673-51-2DP**, **CD44** (antigen) (human clone HUVDE75), fusion protein with IgG Fc fragment **203673-52-3DP**, 1-217-**CD44** (antigen) (human clone HUVDE75), fusion protein with IgG Fc fragment **203743-82-2DP**, 246-301-**CD44** (antigen) (human clone HUVDE75), fusion protein with IgG Fc fragment **237078-02-3DP**, fusion protein with IgG Fc fragment **237078-02-3P**
RL: BPN (Biosynthetic preparation); PRP (Properties); BIOL (Biological study); PREP (Preparation)
(amino acid sequence; protein and DNA sequences **encoding** a human **CD44-like** protein)

IT **203673-47-6 203673-49-8**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(nucleotide sequence of extracellular domain; protein and DNA sequences **encoding** a human **CD44-like** protein)

IT **203673-44-3 203673-45-4 203673-46-5 237078-03-4**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(nucleotide sequence; protein and DNA sequences **encoding** a human **CD44-like** protein)

REFERENCE COUNT: 71 THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:311573 HCAPLUS
DOCUMENT NUMBER: 131:169210
TITLE: Signaling complex formation of CD44 with src-related kinases
AUTHOR(S): Rozsnyay, Zoltan
CORPORATE SOURCE: Department of Tumor Progression and Immune Defense, German Cancer Research Center, Heidelberg, Germany
SOURCE: Immunology Letters (1999), 68(1), 101-108
CODEN: IMLED6; ISSN: 0165-2478
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The complex formation of murine **CD44** with the src-**like** protein tyrosine kinases, lck and lyn, was investigated. In accordance with previous observations, stable **CD44**-lck and **CD44** -lyn complexes were detected in nonstimulated lymphoid T- and B-cells, resp. In addition, a direct modulation of lck and lyn by **CD44** was observed as revealed by the **CD44**-dependent translocation of these enzymes to the Triton X-100 resistant cell fraction. To clarify which receptor domain is responsible for the association, peptide binding assays were performed. Interestingly, the synthetic peptide pCD44 (ILAVCIAVNSRRR), which corresponds to the plasma membrane-cytoplasmic interface region of murine **CD44**, exhibited a high capacity to bind lck and lyn. A single amino acid modification in the position of the cysteine residue completely abolished this interaction, while the truncation of the three tandem arginines significantly decreased it. Remarkably, similar sequences were found in a number of other mols. including subunits of receptors recognizing antigens, Igs, extracellular matrix components, accessory mols., cytokines and also in certain viral gene products. Synthetic peptides corresponding to the **homologous** regions found in CD28 and FcεRIβ were also studied and comparable lck-lyn-binding potentials were detected. These data suggest a novel interaction between src-family kinases and **CD44**, CD28, FcεRIβ, and provide a simple model for the association of src-like kinases with transmembrane proteins.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:126349 HCAPLUS
DOCUMENT NUMBER: 128:189203
TITLE: Cloning and cDNA sequence of human **CD44**-**like** protein and its therapeutic and diagnostic uses
INVENTOR(S): Ni, Jian; Gentz, Reiner L.; Dillon, Patrick J.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA; Ni, Jian; Gentz, Reiner L.; Dillon, Patrick J.
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9806839	A1	19980219	WO 1996-US13008	19960809

W: AM, AU, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IL, JP, KG, KP,

KR, KZ, LT, LV, MD, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TR, UA, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
AU 9667216 A1 19980306 AU 1996-67216 19960809
EP 960198 A1 19991201 EP 1996-927373 19960809
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
PRIORITY APPLN. INFO.: WO 1996-US13008 19960809

AB The present invention concerns a novel **CD44-like** protein receptor. In particular, isolated cDNA **encoding** the **CD44-like** protein was isolated and sequenced from human umbilical vein endothelial cells. The **CD44-like** protein comprises 322 amino acid residues including a 21-residue signal peptide, an extracellular domain (residues 22-238), a transmembrane domain (residues 239-266), and a **CD44-like** protein intracellular domain (residues 267-322). Northern blot anal. detected expression of the gene in most human tissues. **CD44-like** polypeptides are also provided, as are screening methods for identifying agonists and antagonists capable of enhancing or inhibiting **CD44-like** protein-mediated signaling. The invention further concerns therapeutic methods for treating diseases associated with processes mediated by **CD44-like** protein signaling.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:363291 HCAPLUS
DOCUMENT NUMBER: 122:154566
TITLE: Proteoglycan forms of the lymphocyte homing receptor CD44 are alternatively spliced variants containing the v3 exon
AUTHOR(S): Jackson, David G.; Bell, John I.; Dickinson, Richard; Timans, Jackie; Shields, John; Whittle, Nigel
CORPORATE SOURCE: Mol. Immunology Group, Univ. Oxford, Oxford, OX3 9DU, UK
SOURCE: Journal of Cell Biology (1995), 128(4), 673-85
CODEN: JCLBA3; ISSN: 0021-9525
PUBLISHER: Rockefeller University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The **CD44** cell surface glycoprotein is expressed on a broad range of different tissues as multiple isoforms containing from one to ten alternatively spliced exons v1-v10 inserted within the extracellular domain. Differential glycosylation generates still further variability, yielding both N- and O-glycan-modified forms of **CD44** in addition to proteoglycan-**like** variants containing chondroitin sulfate and heparan sulfate. These high mol. mass proteoglycan-like variants, previously identified in lymphocytes, melanomas, and keratinocytes have been implicated in cell-matrix adhesion, cell motility, and invasiveness. More recently, monocyte **CD44** mols. presumed to carry glycosaminoglycan chains were shown to bind the chemokine MIP-1β raising the intriguing possibility that proteoglycan-like **CD44** variants might play a role in regulating inflammatory responses. Here the authors have investigated the mol. identity of these proteoglycan-like **CD44** variants by generating a panel of recombinant **CD44** isoforms using a novel cassette cloning strategy. The authors show that both chondroitin and heparan sulfate modifications are associated specifically with isoforms (CD44v3-10 and CD44v3,8-10) containing the v3 alternative exon which **encodes** a consensus motif SGXG for GAG addition Other isoforms (CD44v10, CD44v8-10, CD44v7-10, and CD44v6-10) are shown to lack these GAG chains but to carry extensive O-glycan modifications, most likely within the mucin-like alternative exon inserts.

The authors also demonstrate that the majority of endogenous GAG-modified **CD44** isoforms present in epithelial cells constitute v3 isoforms thus establishing that in these cells the majority of proteoglycan-like **CD44** variants are generated by alternative splicing. Finally the authors present evidence using transfected B lymphoma cells that the GAG-modified **CD44** isoforms CD44v3-10 and CD44v3,8-10, unlike CD44H, bind only weakly to hyaluronan. Together with the demonstration in the accompanying paper (Bennett, K., D. G. Jackson, J. C. Simon, E. Tanczos, R. Peach, B. Modrell, I. Stamenkovic, G. Plowman, and A. Aruffo. 1995. J. Cell Biol. 128:687-698.), that **CD44** mols. containing the v3 exon bind growth factors, these results highlight a new and potentially important role for **CD44** alternative splicing in the control of cell-surface proteoglycan expression.

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FILE COVERS 1907 - 11 Jul 2004 VOL 141 ISS 3
FILE LAST UPDATED: 9 Jul 2004 (20040709/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> d stat que l29 1-57
'1-57' IS NOT VALID HERE

=> d stat que l29
L1 21 SEA FILE=REGISTRY ABB=ON PLU=ON FASCILIN? OR STABILIN?
L2 379 SEA FILE=REGISTRY ABB=ON PLU=ON CD44 OR CD(L)44
L3 418 SEA FILE=HCAPLUS ABB=ON PLU=ON L1 OR ?FASCILIN? OR ?STABLILIN ?
L4 4329 SEA FILE=HCAPLUS ABB=ON PLU=ON L2 OR CD44 OR CD(W)44
L5 40848 SEA FILE=HCAPLUS ABB=ON PLU=ON FELL
L7 3 SEA FILE=HCAPLUS ABB=ON PLU=ON L3 AND (L4 OR L5)
L10 981 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(L)(?ENCOD? OR CODE? OR CODING OR HOMOLOG?)
L12 42 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(4W)LIKE
L13 6 SEA FILE=HCAPLUS ABB=ON PLU=ON L10 AND L12
L14 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L13 NOT L7
L20 9679 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(L)(EXPRESS? OR ?CLON? OR ?FUSION? OR ?RECOMBIN? OR VECTOR? OR HOST?)

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L23          174 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L3 OR L4 OR L5)(5A)(?DERIVAT?
                 OR ?ANALOG? OR ?FRAGMENT? OR ?HOMOLOG?)
L24           79 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L23 AND L20
L25          240 SEA FILE=REGISTRY ABB=ON  PLU=ON  DEOXYRIBONUCLEIC ACID#/CN OR
                 DNA/CN OR NUCLEIC ACID?/CN
L26         1913 SEA FILE=REGISTRY ABB=ON  PLU=ON  PROTEIN/CN OR PROTEINS
L27         8456 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L3 OR L4 OR L5)(L)(L25 OR
                 ?NUCLEIC(W)ACID OR DNA OR L26 OR PROTEIN OR ?PEPTIDE?)
L28           58 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L24 AND L27
L29           57 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L28 NOT (L7 OR L14)


=> d ibib abs hitrn l29 1-57

L29  ANSWER 1 OF 57  HCAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         2004:252529  HCAPLUS
DOCUMENT NUMBER:          140:286158
TITLE:                    Antibodies to CD44
INVENTOR(S):              Rondon, Isaac J.; Edge, Albert; Baribault, Kent Rachel
PATENT ASSIGNEE(S):       Dyax Corporation, USA; Baribault Kent, Rachel
SOURCE:                   PCT Int. Appl., 128 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.         KIND  DATE           APPLICATION NO.  DATE
     ---------------    ----  --------       ---------------  --------
     WO 2004024750       A2   20040325       WO 2003-US29318  20030915
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA,
             UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC,
             NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ,
             GW, ML, MR, NE, SN, TD, TG
     US 2004110933       A1   20040610       US 2003-663244   20030915
PRIORITY APPLN. INFO.:                    US 2002-410758P  P  20020913
                                          US 2003-469123P  P  20030509
AB   The authors disclose CD44-binding proteins, including
     CD44-binding antibodies and antibody fragments.
     Nucleic acids, recombinant expression
     vectors and host cells for making such proteins
     are also disclosed.  Methods of using the proteins to detect
     CD44 or to modulate a CD44-expressing cell,
     e.g., in a subject, are also described.

L29  ANSWER 2 OF 57  HCAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         2003:778050  HCAPLUS
DOCUMENT NUMBER:          139:291131
TITLE:                    Gene expression profiles in peripheral blood cells in
                          the diagnosis of multiple sclerosis and test kits
                          using marker genes
INVENTOR(S):              Achiron, Anat; Gurevich, Michael; Mandel, Mathilda;
                          Friedman, Nir; Kaminski, Naftali
PATENT ASSIGNEE(S):       Yissum Research Development Company of the Hebrew
                          University of Jerusalem, Israel; Hadasit Medical
                          Research Services and Development Ltd.
```

SOURCE: PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003081201	A2	20031002	WO 2003-IL208	20030313

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2002-365800P P 20020321

AB Markers of multiple sclerosis and methods and kits utilizing same for diagnosing multiple sclerosis in an individual are provided. These markers were identified by microarray anal. of gene expression in peripheral blood monocytes in different stages of the disease in different presentations.

IT **224340-17-4**, **DNA** (human **CD44** antigen gene exon v9 **fragment**) **389195-49-7** **391528-58-8** **392068-89-2**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; gene **expression** profiles in peripheral blood cells in the diagnosis of multiple sclerosis and test kits using marker genes)

L29 ANSWER 3 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:453260 HCAPLUS
DOCUMENT NUMBER: 139:132387
TITLE: 1-40 β-amyloid **protein fragment** modulates the **expression** of **CD44** and CD71 on the astrocytoma cell line in the presence of IL1β and TNFα
AUTHOR(S): Speciale, Livianna; Ruzzante, Stefania; Calabrese, Elena; Saresella, Marina; Taramelli, Donatella; Mariani, Claudio; Bava, Laura; Longhi, Renato; Ferrante, Pasquale
CORPORATE SOURCE: Laboratory of Biology, ONLUS, IRCCS, Milan, Italy
SOURCE: Journal of Cellular Physiology (2003), 196(1), 190-195
CODEN: JCLLAX; ISSN: 0021-9541
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The modulation of **CD44**, VCAM-1 and CD71 **expression** was analyzed by flow cytometry in the 1321N1 astrocytoma cell line in the presence of interleukin-1β (IL1β), tumor necrosis factor-α (TNFα) and 1-40 or 25-35 β-amyloid (Aβ) fragments. The percentage of 1321N1 astrocytoma cell line **expressing** these markers increased significantly after treatment with TNFα or IL1β. The presence of Aβ 1-40 fragment, alone or in combination with IL1β, induced an increase in the percentage of cells **expressing** **CD44**, but not VCAM-1. However, the

concomitant presence of Aβ 1-40 fragment and of IL1β or TNFα caused an increase in the percentage of CD71 pos. cells. In contrast, the shorter Aβ 25-35 fragment was always inactive. These results indicates that Aβ 1-40 fragment, in association with cytokines, can activate this astrocyte-derived cell line and add further elements in favor of the hypothesis that β-amyloid can act as immunol. mediator.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 4 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:243847 HCAPLUS
DOCUMENT NUMBER: 138:380283
TITLE: Porcine SRY promoter is a target for steroidogenic factor 1
AUTHOR(S): Pilon, Nicolas; Daneau, Isabelle; Paradis, Veronique; Hamel, Frederic; Lussier, Jacques G.; Viger, Robert S.; Silversides, David W.
CORPORATE SOURCE: Centre de recherche en reproduction animale, Department of Veterinary Biomedicine, Faculty of Veterinary Medicine, University of Montreal, St-Hyacinthe, QC, J2S 7C6, Can.
SOURCE: Biology of Reproduction (2003), 68(4), 1098-1106
CODEN: BIREBV; ISSN: 0006-3363
PUBLISHER: Society for the Study of Reproduction
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To study the process of mammalian sex determination and in particular to further understand the mechanisms of transcriptional regulation of the SRY gene, we have isolated a 4.5-kilo-base (kb) pig SRY 5' flanking sequence. To facilitate the in vitro anal. of these sequences, we have generated a porcine genital ridge (PGR) cell line (9E11) that **expresses** SRY as well as SOX9, steroidogenic factor-1 (SF-1), and DAX1. Via primer extension anal. on RNA from this cell line, a transcription start site for porcine SRY was identified at -661 base pairs (bps) 5' from the translation initiation site. Deletion studies of the SRY 5' flanking sequences in PGR 9E11 cells demonstrated that -1.4 kb of 5' flanking sequences retained full transcriptional activity compared with the -4.5 kb **fragment**, but that transcriptional activity **fell** when further deletions were made. Sequences down-stream of the transcriptional start site are important for promoter activity, because deleting transcribed but not translated sequences eliminated promoter activity. Sequence anal. of the -1.4 kb fragment identified two potential binding sites for SF-1, at -1369 and at -290 from the ATG. To address the role of SF-1 transactivation in SRY promoter activity, mutagenesis studies of the potential SF-1 binding sites were performed and revealed that these sites were indeed important for SRY promoter activity. Cotransfection studies in a heterologous cell system (mouse CV-1 cells) demonstrated that pig SF-1 was able to transactivate the pig SRY promoter. Gel shift assays confirmed that the upstream site was recognized by mouse SF-1 **protein.** We conclude that two sites for SF-1 transactivation exist within the pig SRY promoter, at -1369 bp and at -290 bp, and that the site at -1369 bp is quant. the most important.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 5 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:173455 HCAPLUS
DOCUMENT NUMBER: 138:198601
TITLE: New drug **recombinant CD44 protein**
INVENTOR(S): Stroemblad, Staffan; Kogerman, Priit; Paell, Taavi
PATENT ASSIGNEE(S): Swed.

SOURCE: PCT Int. Appl., 60 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003018044	A1	20030306	WO 2002-SE1531	20020826

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1418931	A1	20040519	EP 2002-760977	20020826

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

PRIORITY APPLN. INFO.:
SE 2001-2823 A 20010824
US 2001-314971P P 20010824
WO 2002-SE1531 W 20020826

AB **CD44**, the receptor for hyaluronic acid, has complex functions in cellular physiol., cell migration and tumor metastasis. The inventors have previously found that human **CD44** receptor overexpression in mouse fibrosarcoma cells inhibits s.c. tumor growth in mice. Here it is demonstrated that a tumor growth inhibitory effect of **CD44** is caused by block of angiogenesis. Furthermore, the inventors have found that soluble **recombinant CD44** hyaluronic acid binding domain (CD44HABD) inhibits angiogenesis in vivo in cLick and mouse and thereby inhibits human tumor growth of various origins. The anti-angiogenic effect of **CD44**-HABD is independent of hyaluronic acid (HA) binding, since non-HA-binding mutants of CD44HABD still maintain anti-angiogenic properties. The invention discloses soluble **CD44 recombinant proteins** as a novel class of angiogenesis inhibitors based on targeting of vascular cell surface receptor. A method of block of angiogenesis and treatment of human tumors using **recombinant CD44 proteins** as well as their **analogs** is disclosed. As a further embodiment of the invention, methods for screening for new drug targets using **CD44 recombinant proteins** and their **analogs** is presented.

IT **500377-17-3P 500377-24-2P 500377-26-4P 500377-28-6P 500377-30-0P 500377-32-2P 500377-33-3P**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; **recombinant CD44 protein** for antiangiogenic antitumor use)

IT **500377-23-1P 500377-25-3P 500377-27-5P 500377-29-7P 500377-31-1P**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(nucleotide sequence; **recombinant CD44 protein** for antiangiogenic antitumor use)

IT **500377-16-2**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; **recombinant CD44 protein** for antiangiogenic antitumor use)
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 6 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:133311 HCAPLUS
DOCUMENT NUMBER: 138:186409
TITLE: CD44 variants carrying heparan sulfate chains and uses thereof
INVENTOR(S): Yayon, Avner; Nedvetzki, Shlomo; Naor, David; Golan, Itshak
PATENT ASSIGNEE(S): Yissum Research Development Company of the Hebrew University of Jerusalem, Israel
SOURCE: PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003014160	A2	20030220	WO 2002-IL653	20020808
WO 2003014160	A3	20031016		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-310840P P 20010809
AB Modulation of the activity of a heparin-binding growth factor (HBGF) by enhancing or inhibiting high affinity binding of said HBGF to its receptor, can be achieved with an agent selected from: (i) a soluble **CD44** isoform carrying at least one chain of a heparan sulfate; (ii) a **recombinant** chimeric **fusion protein** comprising the amino acid sequence of a soluble **CD44** isoform fused to a tag suitable for proteoglycan purification, said **fusion** mol. being post-translationally glycosylated to carry at least one chain of a heparan sulfate; and (iii) a sugar mol. being a heparan sulfate derived from a **CD44** isoform, or a **fragment** thereof. The agents (i) and (ii) when the soluble **CD44** isoform is the soluble **CD44** variant **expressed** in synovial cells of rheumatoid arthritis patients (CD44vRA), and the heparan sulfate of (iii), are novel.
IT **62683-29-8**, Csf
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**CD44** variants carrying heparan sulfate chains and uses thereof in rheumatoid arthritis)

L29 ANSWER 7 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:885978 HCAPLUS
DOCUMENT NUMBER: 137:389130
TITLE: Conjugates of an antibody to CD44 and a maytansinoid

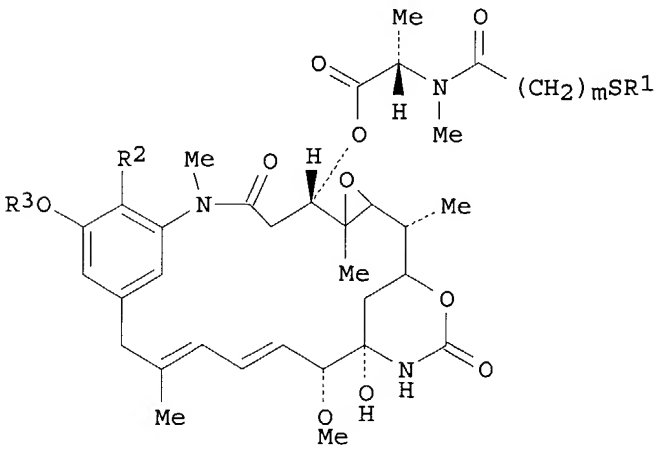
INVENTOR(S): Adolf, Guenther; Heider, Karl-Heinz; Patzelt, Erik; Sproll, Marlies
PATENT ASSIGNEE(S): Boehringer Ingelheim International GmbH, Germany
SOURCE: Eur. Pat. Appl., 31 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1258255	A1	20021120	EP 2001-112227	20010518
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
WO 2002094325	A2	20021128	WO 2002-EP5413	20020516
WO 2002094325	A3	20030417		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1395290	A2	20040310	EP 2002-753054	20020516
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
EE 200300568	A	20040415	EE 2003-568	20020516
BR 2002009862	A	20040608	BR 2002-9862	20020516
US 2003103985	A1	20030605	US 2002-150475	20020517
NO 2003005108	A	20031117	NO 2003-5108	20031117

PRIORITY APPLN. INFO.: EP 2001-112227 A 20010518
US 2001-307451P P 20010724
WO 2002-EP5413 W 20020516

OTHER SOURCE(S): MARPAT 137:389130

GI



I

AB The present invention relates to novel conjugates of antibodies with cytotoxic compds., pharmaceutical compns. containing such conjugates, and their use in cancer therapy. In particular, the present invention relates

to conjugates of antibodies which are specific for CD44 with maytansinoids, preferably with N2'-deacetyl-N2'-(3-mercapto-1-oxopropyl)-maytansine. In a particularly preferred embodiment, the antibody/maytansinoid conjugate may be prepared from a maytansinoid of formula (I) wherein R1 represents H or SR4, wherein R4 represents Me, Et, linear alkyl, branched alkyl, cyclic alkyl, simple or substituted aryl, or heterocyclic; R2 represents Cl or H; R3 represents H or CH3; and m represents 1,2, or 3. Preferably, R1 is H or CH3, R2 is Cl, R3 is CH3, and m = 2. The compound with R1 = H, R2 = Cl, R3 = CH3, and m = 2 is designated DM1 in the literature.

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 8 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:862070 HCAPLUS
DOCUMENT NUMBER: 138:104736
TITLE: **CD44** stimulation by **fragmented** hyaluronic acid induces upregulation of urokinase-type plasminogen activator and its receptor and subsequently facilitates invasion of human chondrosarcoma cells
AUTHOR(S): Kobayashi, Hiroshi; Suzuki, Mika; Kanayama, Naohiro; Nishida, Takashi; Takigawa, Masaharu; Terao, Toshihiko
CORPORATE SOURCE: Department of Obstetrics and Gynecology, Hamamatsu University School of Medicine, Shizuoka, 431-3192, Japan
SOURCE: International Journal of Cancer (2002), 102(4), 379-389
CODEN: IJCNAW; ISSN: 0020-7136
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB It has been established that fragmented hyaluronic acid (HA), but not native high mol. weight HA, can induce angiogenesis, cell proliferation and migration. We have studied the outside-in signal transduction pathways responsible for fragmented HA-mediated cancer cell invasion. In our study, we have studied the effects of **CD44** stimulation by ligation with HA upon the **expression** of matrix metalloproteinases (MMPs)-2 and -9 as well as urokinase-type plasminogen activator (uPA), its receptor (uPAR) and its inhibitor (PAI-1) and the subsequent induction of invasion of human chondrosarcoma cell line HCS-2/8. Our study indicates that (i) **CD44** stimulation by **fragmented** HA upregulates **expression** of uPA and uPAR mRNA and **protein** but does not affect MMPs secretion or PAI-1 mRNA **expression**; (ii) the effects of HA fragments are critically HA size dependent: high mol. weight HA is inactive, but lower mol. weight fragmented HA (Mr 3.5 kDa) is active; (iii) cells can bind avidly Mr 3.5 kDa **fragmented** HA through a **CD44** mol., whereas cells do not effectively bind higher Mr HA; (iv) a fragmented HA induces phosphorylation of MAP kinase **proteins** (MEK1/2, ERK1/2 and c-Jun) within 30 min; (v) **CD44** is critical for the response (activation of MAP kinase and upregulation of uPA and uPAR **expression**); and (vi) cell invasion induced by **CD44** stimulation with a **fragmented** HA is inhibited by anti-**CD44** mAb, MAP kinase inhibitors, neutralizing anti-uPAR pAb, anti-catalytic anti-uPA mAb or amiloride. Therefore, our study represents the first report that **CD44** stimulation induced by a **fragmented** HA results in activation of MAP kinase and, subsequently, enhances uPA and uPAR **expression** and facilitates invasion of human chondrosarcoma cells.

IT **137632-07-6**, ERK1 kinase **137632-08-7**, ERK2 kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(**CD44** stimulation by **fragmented** hyaluronic acid induces phosphorylation of MAP kinases and subsequently facilitates invasion of human chondrosarcoma cells)

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 9 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:634856 HCAPLUS
DOCUMENT NUMBER: 137:215686
TITLE: **CD44** stimulation by **fragmented** hyaluronic acid induces upregulation and tyrosine phosphorylation of c-Met receptor **protein** in human chondrosarcoma cells
AUTHOR(S): Suzuki, Mika; Kobayashi, Hiroshi; Kanayama, Naohiro; Nishida, Takashi; Takigawa, Masaharu; Terao, Toshihiko
CORPORATE SOURCE: Department of Obstetrics and Gynecology, Hamamatsu University School of Medicine, Hamamatsu, Shizuoka, 431-3192, Japan
SOURCE: Biochimica et Biophysica Acta (2002), 1591(1-3), 37-44
CODEN: BBACAQ; ISSN: 0006-3002
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Hepatocyte growth factor/scatter factor (HGF/SF) can induce proliferation and motility and promote invasion of tumor cells. Since HGF/SF receptor, c-Met, is **expressed** by tumor cells, and since stimulation of **CD44**, a transmembrane glycoprotein known to bind hyaluronic acid (HA) in its extracellular domain, is involved in activation of c-Met, we have studied the effects of **CD44** stimulation by ligation with HA upon the **expression** and tyrosine phosphorylation of c-Met on human chondrosarcoma cell line HCS-2/8. The current study indicates that (a) **CD44** stimulation by **fragmented** HA upregulates **expression** of c-Met **proteins**; (b) fragmented HA also induces tyrosine phosphorylation of c-Met **protein** within 30 min, an early event in this pathway as shown by the early time course of stimulation; (c) the effects of HA fragments are critically HA size-dependent. High mol. weight HA is inactive, but lower mol. weight fragments (Mr 3.5 kDa) are active with maximal effect in the μg/mL range; (d) the standard form of **CD44** (CD44s) is critical for the response because the effect on c-Met, both in terms of upregulation and phosphorylation, is inhibited by preincubation with an anti-**CD44** **monoclonal** antibody; and (e) phosphorylation of c-Met induced by **CD44** stimulation is inhibited by **protein** tyrosine kinase inhibitor, tyrphostin. Therefore, our study represents the first report that **CD44** stimulation induced by **fragmented** HA enhances c-Met **expression** and tyrosine phosphorylation in human chondrosarcoma cells. These studies establish a signal transduction cascade or cross-talk emanating from **CD44** to c-Met.
REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 10 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:556908 HCAPLUS
DOCUMENT NUMBER: 137:274705
TITLE: Hyaluronan binding properties of a CD44 chimera containing the link module of TSG-6
AUTHOR(S): Lesley, Jayne; English, Nicole M.; Gal, Istvan; Mikecz, Katalin; Day, Anthony J.; Hyman, Robert
CORPORATE SOURCE: Molecular and Cell Biology Laboratory, Salk Institute, San Diego, CA, 92186, USA
SOURCE: Journal of Biological Chemistry (2002), 277(29), 26600-26608

CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **CD44**, a cell-surface receptor for the extracellular matrix glycosaminoglycan hyaluronan, can mediate leukocyte rolling on hyaluronan substrates and has been implicated in leukocyte migration to sites of inflammation. **CD44**-mediated binding to hyaluronan is of low affinity, and effective cell/matrix interaction depends on multiple interactions with the multivalent ligand. We replaced the Link module of **CD44** with the **homologous** region of TSG-6, a hyaluronan-binding **protein** secreted in response to inflammation whose Link module has a higher affinity for ligand. **Monoclonal** antibodies raised against the **CD44**/TSG-6 chimera recognized **recombinant** human TSG-6 and native mouse TSG-6 and blocked hyaluronan binding to these **proteins.** Cells **expressing** the **CD44**/TSG-6 mol. bound hyaluronan with higher avidity than cells **expressing CD44.** This resulted in changes in the hyaluronan binding properties characteristic of cells **expressing CD44** such as requirements for threshold levels of receptor **expression** and for hyaluronan of high mol. mass. In parallel plate flow assays used to model leukocyte rolling, cells **expressing CD44**/TSG-6 failed to roll on hyaluronan. Instead, they stuck and remained "tethered" to the substrate under fluid flow. This result argues that the low affinity of **CD44** for its ligand is important for rolling, an early phase of leukocyte extravasation from the blood.

REFERENCE COUNT: 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 11 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:102080 HCAPLUS
DOCUMENT NUMBER: 136:323624

TITLE: Characterization of Endogenous Chinese Hamster Ovary Cell Surface Molecules That Mediate T Cell Costimulation

AUTHOR(S): Gaglia, Jason L.; Mattoo, Aditya; Greenfield, Edward A.; Freeman, Gordon J.; Kuchroo, Vijay K.
CORPORATE SOURCE: Center For Neurologic Diseases, Department of Neurology, Brigham & Women's Hospital and Harvard Medical School, Boston, MA, 02115, USA

SOURCE: Cellular Immunology (2001), 213(2), 83-93
CODEN: CLIMB8; ISSN: 0008-8749
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Chinese hamster ovary (CHO) cells are commonly used in the generation of transfectants for use in in vitro costimulation assays. However, the authors have noted that nontransfected CHO cells can themselves provide a low-level B7/CD28 independent costimulatory signal for CD3-mediated murine T cell activation and IL-2 production This study set out to identify those mols. that contribute to this CHO-dependent costimulatory activity. The authors describe a CHO subline capable of delivering potent CD28-independent costimulation to murine T cells and the generation of **monoclonal** antibodies against these CHO cells that inhibited this costimulatory activity. These blocking antibodies do not affect CHO cell-independent costimulation or bind mouse cells, suggesting an effect mediated by their target mols. on the costimulatory competent CHO cells. Immunopptn. and **expression cloning** revealed that these antibodies bound the hamster **homologs** of Crry (CD21/35), **CD44**, CD54 (ICAM-1), CD63, CD87, CD147, and an 80- to 90-kDa

protein which could not be **cloned. Expression** of these hamster genes on COS cells demonstrated that hamster CD54 was able to costimulate both CD3-mediated IL-2 secretion and T cell proliferation by naive murine T cells independent of the other mols. identified. (c) 2001 Academic Press.

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 12 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:886495 HCAPLUS
DOCUMENT NUMBER: 136:35876
TITLE: Membrane-type 1 matrix metalloproteinase cleavage of CD44 as indicator of cell migration, infiltration, and metastasis
INVENTOR(S): Seiki, Motoharu; Obata, Ken-ichi; Oku, Tohru
PATENT ASSIGNEE(S): Fujichemico, Ltd., Japan
SOURCE: PCT Int. Appl., 206 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001092507	A1	20011206	WO 2001-JP4567	20010530

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 2000-159533 A 20000530

AB **CD44 fragments** resulting from cleavage by matrix metalloproteinase (MMP) and their use in detecting or measuring the migration, infiltration, wandering and/or metastasis of cells relating to pathol. conditions (for example, cancer cell metastasis, blood cell infiltration) is disclosed. Moreover, **recombinant expression** and use of mutant **CD44 proteins** resistant to MMP cleavage, MMP inhibitors, anti-MMP antibodies, etc. for screening of anticancer agents, antimetastasis agents, antiinflammatory agents, and immune disorder drugs, is claimed. It was found that shedding of **CD44** by MMPs (in particular membrane-attached MMP such as MT1-MMP) triggers morphol. and functional changes of cells. Based on this finding, it is possible to provide a means of estimating pathol. conditions such as cancer cell metastasis and blood cell infiltration by assaying **CD44 fragments** formed by the shedding and reagents such as **monoclonal** antibodies. Migratory cells including invasive tumor cells frequently **express CD44**, a major receptor for hyaluronan and membrane-type 1 matrix metalloproteinase (MT1-MMP) that degrades extracellular matrix at the pericellular region. In this study, we demonstrate that MT1-MMP acts as a processing enzyme for CD44H, releasing it into the medium as a soluble 70-kD fragment. Furthermore, this processing event stimulates cell motility; however, **expression** of either CD44H or MT1-MMP alone did not stimulate cell motility. Coexpression of MT1-MMP and mutant CD44H lacking the MT1-MMP-processing site did not result in shedding and did not promote cell migration, suggesting that the processing of CD44H by MT1-MMP is critical in the migratory stimulation. Moreover, **expression** of the mutant CD44H

inhibited the cell migration promoted by CD44H and MT1-MMP in a dominant-neg. manner. The pancreatic tumor cell line, MIA PaCa-2, was found to shed the 70-kD CD44H fragment in a MT1-MMP-dependent manner. **Expression** of the mutant CD44H in the cells as well as MMP inhibitor treatment effectively inhibited the migration, suggesting that MIA PaCa-2 cells indeed use the CD44H and MT1-MMP as migratory devices. These findings revealed a novel interaction of the two mols. that have each been implicated in tumor cell migration and invasion.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 13 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:879693 HCAPLUS
DOCUMENT NUMBER: 136:115875
TITLE: Proteolytic release of CD44 intracellular domain and its role in the CD44 signaling pathway
AUTHOR(S): Okamoto, Isamu; Kawano, Yoshiaki; Murakami, Daizo; Sasayama, Takashi; Araki, Norie; Miki, Toru; Wong, Albert J.; Saya, Hideyuki
CORPORATE SOURCE: Department of Tumor Genetics and Biology, Kumamoto University School of Medicine, Kumamoto, 860-0811, Japan
SOURCE: Journal of Cell Biology (2001), 155(5), 755-762
CODEN: JCLBA3; ISSN: 0021-9525
PUBLISHER: Rockefeller University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **CD44** is a widely distributed cell surface adhesion mol. and is implicated in diverse biol. processes. However, the nature of intracellular signaling triggered by **CD44** remains to be elucidated. Here, we show that **CD44** undergoes sequential proteolytic cleavage in the ectodomain and intracellular domain, resulting in the release of a **CD44** intracellular domain (ICD) **fragment.** Consequently, CD44ICD acts as a signal transduction mol., where it translocates to the nucleus and activates transcription mediated through the 12-O-tetradecanoylphorbol 13-acetate-responsive element, which is found in numerous genes involved in diverse cellular processes. **Expression** of an uncleavable **CD44** mutant as well as metalloprotease inhibitor treatment blocks **CD44** -mediated transcriptional activation. In search of the underlying mechanism, we have found that CD44ICD potentiates transactivation mediated by the transcriptional coactivator CBP/p300. Furthermore, we show that cells **expressing** CD44ICD produce high levels of **CD44** mRNA, suggesting that the **CD44** gene is one of the potential targets for transcriptional activation by CD44ICD. These observations establish a novel **CD44** signaling pathway and shed new light on the functional link between proteolytic processing of an adhesion mol. at the cell surface and transcriptional activation in the nucleus.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 14 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:780962 HCAPLUS
DOCUMENT NUMBER: 135:340221
TITLE: Differentially expressed nucleic acids and their encoded proteins for the therapy and diagnosis of human breast cancer
INVENTOR(S): Jiang, Yuqiu; Dillon, Davin C.; Mitcham, Jennifer L.; Xu, Jiangchun; Harlocker, Susan L.; Hepler, William T.
PATENT ASSIGNEE(S): Corixa Corporation, USA
SOURCE: PCT Int. Appl., 297 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 16
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001079286	A2	20011025	WO 2001-US12164	20010412
WO 2001079286	A3	20030130		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2003104366	A1	20030605	US 2000-551621	20000417
US 6756477	B1	20040629	US 2000-590751	20000608
US 2002064872	A1	20020530	US 2000-604287	20000622
US 6586572	B2	20030701		
US 6528054	B1	20030304	US 2000-620405	20000720
AU 2001055369	A5	20011030	AU 2001-55369	20010412
EP 1299417	A2	20030409	EP 2001-928521	20010412
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001010091	A	20040225	BR 2001-10091	20010412
NO 2002004972	A	20021211	NO 2002-4972	20021016

PRIORITY APPLN. INFO.:

US 2000-551621	A	20000417
US 2000-590751	A	20000608
US 2000-604287	A	20000622
US 2000-620405	A	20000720
US 1998-222575	A2	19981228
US 1999-285480	A2	19990402
US 1999-339338	A2	19990623
US 1999-389681	A2	19990902
US 1999-433826	A2	19991103
WO 2001-US12164	W	20010412

AB Compns. and methods for the therapy and diagnosis of cancer, such as breast cancer, are disclosed. Compns. may comprise one or more breast tumor proteins, immunogenic portions thereof, or polynucleotides that encode such portions. PCR-based cDNA library subtraction was used to identify transcripts and their encoded proteins that are differentially expressed in breast tumor tissues in comparison to normal breast tissue. A therapeutic composition may also comprise an antigen-presenting cell that expresses a breast tumor protein, or a T cell that is specific for cells expressing such a protein. Such compns. may be used, for example, for the prevention and treatment of diseases such as breast cancer. Diagnostic methods based on detecting a breast tumor protein, or mRNA encoding such a protein, in a sample are also provided.

L29 ANSWER 15 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:168146 HCAPLUS
DOCUMENT NUMBER: 134:202678
TITLE: Sequences of human genes involving in HIV replication and uses thereof in therapy and drug screening
INVENTOR(S): Holzmayer, Tanya A.; Dunn, Stephen J.
PATENT ASSIGNEE(S): Subsidiary No. 3, Inc., USA
SOURCE: PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001016322	A2	20010308	WO 2000-US24262	20000901
WO 2001016322	A3	20020711		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1238072	A2	20020911	EP 2000-961525	20000901
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003510033	T2	20030318	JP 2001-520868	20000901

PRIORITY APPLN. INFO.: US 1999-388182 A1 19990901
WO 2000-US24262 W 20000901

AB The present invention relates to the identification of a number of human genes as cellular targets for the design of therapeutic agents for suppressing human immunodeficiency virus (HIV) infection. These genes encode products which appear to be necessary for HIV replication, as evidenced by an inhibition of HIV infection in cells in which the expression of these genes is down-regulated. In addition, the invention also relates to methods for identifying addnl. cellular genes as therapeutic targets for suppressing HIV infection, and methods of using such cellular genes and their encoded products in screening assays for selecting addnl. inhibitors of HIV. Thus, two selection strategies were used to isolate human cell-derived genetic suppressor elements (GSEs) with HIV-suppressive activities. One strategy selected for GSEs which suppressed productive infection of cells by HIV. The second strategy selected for GSEs which suppressed induction of the latent provirus in OM10.1 cells. Twenty one cDNAs were identified from RFE library made from CEM-ss cells. Another fourteen GSEs were isolated from RFL library made from peripheral blood mononuclear cells (PBMC). Thirteen GSEs were demonstrated to be able to inhibit translocation of the HIV protein Rev.

IT **217306-82-6**, **DNA** (human **clone** CF-302 **CD44** (antigen) **fragment**-specifying cDNA)
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(nucleotide sequence; sequences of human genes involving in HIV replication and uses thereof in therapy and drug screening)

L29 ANSWER 16 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:81189 HCAPLUS
DOCUMENT NUMBER: 132:220474
TITLE: CD44 interaction with Tiam1 promotes Rac1 signaling and hyaluronic acid-mediated breast tumor cell migration
AUTHOR(S): Bourguignon, Lilly Y. W.; Zhu, Hongbo; Shao, Lijun; Chen, You Wei
CORPORATE SOURCE: Department of Cell Biology and Anatomy, School of Medicine, University of Miami, Miami, FL, 33101, USA
SOURCE: Journal of Biological Chemistry (2000), 275(3), 1829-1838
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular

Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In this study we have explored the interaction between **CD44** (the hyaluronic acid (HA)-binding receptor) and Tiam1 (a guanine nucleotide exchange factor) in metastatic breast tumor cells (SP1 cell line). Immunopptn. and immunoblot analyses indicate that both the CD44v3 isoform and the Tiam1 **protein** are **expressed** in SP1 cells and that these two **proteins** are phys. associated as a complex in vivo. Using an Escherichia coli-derived calmodulin-binding **peptide** -tagged Tiam1 fragment (i.e. the NH2-terminal pleckstrin homol. (PHn) domain and an adjacent **protein** interaction domain designated as PHn-CC-Ex, amino acids 393-738 of Tiam1) and an in vitro binding assay, we have detected a specific binding interaction between the Tiam1 PHn-CC-Ex domain and **CD44.** Scatchard plot anal. indicates that there is a single high affinity **CD44** binding site in the PHn-CC-Ex domain of Tiam1 with an apparent dissociation constant (Kd) of 0.2 nM, which is comparable with **CD44** binding (Kd = .apprx.0.13 nM) to intact Tiam1. These findings suggest that the PHn-CC-Ex domain is the primary Tiam1-binding region for **CD44.** Most importantly, the binding of HA to CD44v3 of SP1 cells stimulates Tiam1-catalyzed Rac1 signaling and cytoskeleton-mediated tumor cell migration. Transfection of SP1 cells with Tiam1 cDNA promotes Tiam1 association with CD44v3 and up-regulates Rac1 signaling as well as HA/CD44v3-mediated breast tumor cell migration. Co-transfection of SP1 cells with PHn-CC-Ex cDNA and Tiam1 cDNA effectively inhibits Tiam1 association with **CD44** and efficiently blocks tumor behaviors. Apparently, the linkage between CD44v3 isoform and the PHn-CC-EX domain of Tiam1 is required for HA stimulated Rac1 signaling and cytoskeleton-mediated tumor cell migration during breast cancer progression.

REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 17 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:580532 HCAPLUS
DOCUMENT NUMBER: 131:309381
TITLE: CD44 stimulation induces integrin-mediated adhesion of colon cancer cell lines to endothelial cells by up-regulation of integrins and c-met and activation of integrins
AUTHOR(S): Fujisaki, Takeshi; Tanaka, Yoshiya; Fujii, Koichi; Mine, Shinichiro; Saito, Kazuyoshi; Yamada, Shinwa; Yamashita, Uki; Irimura, Tatsuro; Eto, Sumiya
CORPORATE SOURCE: The First Department of Internal Medicine, School of Medicine, University of Occupational and Environmental Health, Japan, Kitakyushu, 807-8555, Japan
SOURCE: Cancer Research (1999), 59(17), 4427-4434
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: AACR Subscription Office
DOCUMENT TYPE: Journal
LANGUAGE: English

AB For cancer metastasis, tumor cells present in the circulation must first adhere to the endothelium. Integrins lymphocyte function-associated antigen (LFA) 1 and very late antigen 4 play a central role in leukocyte adhesion to the endothelium and subsequent migration into tissues. The majority of tumor cells derived from solid cancers including colorectal cancer do not **express** suitable adhesion receptors, LFA-1 and very late antigen 4. We investigated the mechanisms of adhesion and transendothelial migration of cancer cells using colorectal carcinoma cell lines. Our results showed the following novel features of **CD44** on the cells: (a) colon cancer cells **express** high levels of **CD44**; (b) stimulation of cancer cells by **CD44**

crosslinking or **fragmented** hyaluronan markedly induces the **expression** of LFA-1s, some of which reveal an activation epitope on the cells; (c) **CD44** crosslinking induces F-actin polymerization in the cell cortex; (d) fragmented hyaluronan induces up-regulation of the activation epitope of LFA-1, which is mediated through **protein** kinase C; (e) stimulation of **CD44** augments the LFA-1-mediated adhesion of cancer cells to endothelial cells and intercellular adhesion mol. 1-transfected cells and facilitates transendothelial migration; (f) stimulation of **CD44** also induces **expression** of the hepatocyte growth factor (HGF) receptor c-Met on cancer cells; and (g) HGF further amplifies the LFA-1-mediated adhesion of cells prestimulated by **CD44**-derived signaling. Our results indicated that stimulation by **CD44** induces "outside-in signaling," which consists of a direct pathway via **CD44** and an alternate pathway through the induction of c-Met **expression** via HGF. Such stimuli augment the **expression** and trigger the function of integrins via "inside-out signaling" in colon cancer cells, which leads to amplification of integrin-mediated adhesion to the vessel wall and subsequent transendothelial migration.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 18 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:88596 HCAPLUS
DOCUMENT NUMBER: 130:266105
TITLE: Characterization of the heparan sulfate and chondroitin sulfate assembly sites in CD44
AUTHOR(S): Greenfield, Brad; Wang, Wei-Chun; Marquardt, Hans; Piepkorn, Michael; Wolff, Edith A.; Aruffo, Alejandro; Bennett, Kelly L.
CORPORATE SOURCE: Bristol-Myers Squibb Pharmaceutical Research Institute, Princeton, NJ, 08543, USA
SOURCE: Journal of Biological Chemistry (1999), 274(4), 2511-2517
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Isoforms of **CD44** are differentially modified by the glycosaminoglycans (GAGs) chondroitin sulfate (CS), heparan sulfate (HS), and keratan sulfate. GAG assembly occurs at serines followed by glycines (SG), but not all SG are utilized. Seven SG motifs are distributed in five **CD44** exons, and in this paper the authors identify the HS and CS assembly sites that are utilized in **CD44**. Not all the **CD44** SG sites are modified. The SGSG motif in **CD44** exon V3 is the only HS assembly site; this site is also modified with CS. HS and CS attachment at that site was eliminated by mutation of the serines in the V3 motif to alanine (AGAG). Exon E5 is the only other **CD44** exon that supports GAG assembly and is modified with CS. Using a number of **recombinant CD44 protein fragments** the authors show herein that the eight amino acids located downstream of the SGSG site in V3 are responsible for the specific addition of HS to this site. If the eight amino acids located downstream from the first SG site in **CD44** exon E5 are exchanged with those located downstream of the SGSG site in exon V3, the SG site in E5 becomes modified with HS and CS. Likewise if the eight amino acids found downstream from the first SG in E5 are placed downstream from the SGSG in V3, this site is modified with CS but not HS. The authors also show that these sequences cannot direct the modification of **CD44** with HS from a distance. Constructs containing **CD44** exon V3 in which the SGSG motif was mutated to AGAG were not modified with HS even though they contained other

SG motifs. Thus, a number of sequence and structural requirements that dictate GAG synthesis on **CD44** have been identified.
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 19 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:17361 HCAPLUS
DOCUMENT NUMBER: 130:219353
TITLE: Cloning genes responsive to a hepatocarcinogenic peroxisome proliferator chemical reveal novel targets of regulation
AUTHOR(S): Corton, J. Christopher; Moreno, Evelyn S.; Merritt, Angel; Bocos, Carlos; Cattley, Russell C.
CORPORATE SOURCE: Chemical Industry Institute of Toxicology, Research Triangle Park, NC, 27709-2137, USA
SOURCE: Cancer Letters (Shannon, Ireland) (1998), 134(1), 61-71
CODEN: CALEDQ; ISSN: 0304-3835
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To better understand the mol. basis of the hepatocyte proliferation and induction of hepatocellular adenomas by exposure to peroxisome proliferator chems. (PPC), a systematic search for genes modulated by a PPC (WY-14643) in rat liver was carried out using the differential display technique. The **fragments fell** into two classes based on the time of initial and maximal induction by WY-14643. The class I genes (**clones** 5 and 30) were induced 3 h after a gavage exposure to WY-14643 with maximal **expression** at 24 h. The class II genes (**clones** 13 and 16) were induced after 24 h with maximal **expression** at 78 wk. **Expression** of the class II genes was also increased after other treatments that cause cell proliferation. **Clone** 30 was identified as CYP4A2, previously shown to be regulated by PPC. **Clone** 13 was homologous to the mouse **protein** H gene, a component of the heterogeneous nuclear ribonucleoprotein particle important in mRNA splicing. **Clone** 16 was identified as cyclophilin-A, the receptor for the immunosuppressant drug cyclosporin A. The sequence of **clone** 5 was unique. These data demonstrate that WY-14643 increases the levels of a number of novel genes that are coordinately regulated with increases in chronic cell proliferation and fatty acid metabolism
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 20 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:541365 HCAPLUS
DOCUMENT NUMBER: 129:274579
TITLE: Regulation of **CD44** gene **expression** by the proinflammatory cytokine interleukin-1β in vascular smooth muscle cells
AUTHOR(S): Foster, Lauren C.; Arkonac, Burak M.; Sibinga, Nicholas E. S.; Shi, Chengwei; Perrella, Mark A.; Haber, Edgar
CORPORATE SOURCE: Cardiovascular Biology Laboratory, Harvard School of Public Health, Boston, MA, 02115, USA
SOURCE: Journal of Biological Chemistry (1998), 273(32), 20341-20346
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The **CD44** gene codes for a family of alternatively spliced, multifunctional adhesion mols. that participate in extracellular matrix binding, lymphocyte activation, cell migration, and tumor metastasis. In a mouse model of transplant-associated arteriosclerosis, **CD44 protein** was induced in the neointima of allografted vessels and colocalized with a subset of proliferating vascular smooth muscle cells (SMC). To elucidate the mol. mechanisms regulating **CD44 expression** in this model, the authors investigated the regulation of **CD44** gene **expression** by interleukin (IL)-1β. Treatment of rat aortic SMC with IL-1β resulted in a 5.3-fold increase in cell surface **CD44 expression.** Northern anal. showed that IL-1β promoted a dose- and time-dependent induction of **CD44** mRNA which reached 6.6-fold after 48 h, and nuclear run-on anal. showed that IL-1β increased the rate of **CD44** gene transcription within 8 h of stimulation. In transient reporter gene transfection expts. in rat aortic SMC, a 1.4-kilobase **fragment** of the mouse **CD44** 5'-flanking sequence mediated this response to IL-1β. Regulation of **CD44** gene **expression** by the proinflammatory cytokine IL-1β may contribute to SMC phenotypic modulation in the pathogenesis of arteriosclerosis.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 21 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:618305 HCAPLUS
DOCUMENT NUMBER: 127:245181
TITLE: Determination of hyaluronic acid using CD44 and kit for determination
INVENTOR(S): Miyaura, Shuichi; Ishimaru, Takeshi
PATENT ASSIGNEE(S): Seikagaku Kogyo Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 16 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 09229930	A2	19970905	JP 1996-32022	19960220

PRIORITY APPLN. INFO.: JP 1996-32022 19960220

AB A method for the determination of hyaluronic acid (I) contains treatment of **CD44** with I in test samples and optionally I-binding **proteins** to form their complexes. **CD44** may be partial **proteins** or fused **proteins** having the I-binding region of **CD44.** The kit for the determination of I contain **CD44.** The kit is useful for diagnosis of rheumatoid arthritis, cancer, liver diseases, etc., when blood concentration of I is increased. A series of standard solns. of I were incubated in a well plate which was previously coated with a phosphate buffer solution containing a fused **protein** of an extracellular domain of **CD44** and human IgG1 Fc **fragment** at 37° for 60 min and treated with biotin-labeled I-binding **protein** and peroxidase-labeled streptavidin at 37° for 60 min. After stopping the reaction, tetramethylbenzidine was added and absorbance of the sandwiched product in each cell was measured. The lower detection limit was 0.1 ng/mL. I was also determined by a competitive method.

L29 ANSWER 22 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:465028 HCAPLUS
DOCUMENT NUMBER: 127:79784
TITLE: Diagnosis or therapy of epithelial carcinoma based on CD44 gene variant exon v6 and encoded antigen fragment

using antibodies
INVENTOR(S): Heider, Karl-Heinz; Adolf, Guenther; Ostermann, Elinborg; Patzelt, Erik; Sproll, Marlies
PATENT ASSIGNEE(S): Boehringer Ingelheim International Gmbh, Germany
SOURCE: Ger. Offen., 13 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 19545472	A1	19970612	DE 1995-19545472	19951206
ZA 9610183	A	19970606	ZA 1996-10183	19961204
CA 2239709	AA	19970612	CA 1996-2239709	19961205
WO 9721104	A1	19970612	WO 1996-EP5448	19961205
W: AU, BG, BR, BY, CA, CN, CZ, EE, HU, IL, JP, KR, KZ, LT, LV, MX, NO, NZ, PL, RO, RU, SG, SK, TR, UA, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9711773	A1	19970627	AU 1997-11773	19961205
AU 726704	B2	20001116		
EP 865609	A1	19980923	EP 1996-942362	19961205
EP 865609	B1	20030319		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, LT, LV, FI				
CN 1207811	A	19990210	CN 1996-199248	19961205
BR 9611901	A	19990302	BR 1996-11901	19961205
JP 2000502067	T2	20000222	JP 1997-520993	19961205
NZ 324314	A	20000228	NZ 1996-324314	19961205
EE 3783	B1	20020617	EE 1998-164	19961205
RU 2193779	C2	20021127	RU 1998-112600	19961205
PL 184521	B1	20021129	PL 1996-327066	19961205
AT 235056	E	20030415	AT 1996-942362	19961205
ES 2190484	T3	20030801	ES 1996-942362	19961205
PT 865609	T	20030829	PT 1996-942362	19961205
NO 9802588	A	19980805	NO 1998-2588	19980605
BG 62985	B1	20001229	BG 1998-102513	19980605
HK 1011560	A1	20031121	HK 1998-112910	19981207

PRIORITY APPLN. INFO.: DE 1995-19545472 A 19951206
DE 1996-19615074 A 19960417
WO 1996-EP5448 W 19961205

AB A method for diagnosis and therapy of epithelial carcinomas is disclosed that is based on the **CD44** antigen **fragment expressed** by the gene variable exon v6. Immunol. determination of the variant antigen fragment using antibody probes is included. Especially useful is the **monoclonal** antibody BIWA-1 (VFF-18). Immunotherapy using antibodies is also claimed.

L29 ANSWER 23 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:386396 HCAPLUS
DOCUMENT NUMBER: 127:46720
TITLE: Protein evolution viewed through Escherichia coli protein sequences: introducing the notion of a structural segment of homology, the module
AUTHOR(S): Riley, Monica; Labedan, Bernard
CORPORATE SOURCE: Marine Biological Lab., Woods Hole, MA, 02543, USA
SOURCE: Journal of Molecular Biology (1997), 268(5), 857-868
CODEN: JMOBAK; ISSN: 0022-2836
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Paralogous genes are genes which descend from a progenitor gene which has duplicated as an ancestral gene, each copy having diverged prior to speciation. With comprehensive information available on functions of E. coli **proteins**, anal. of sequence-related E. coli paralogous **proteins** can give information on the early ancestors of families of **proteins** now residing in many contemporary organisms, such as the enzymes of metabolism, some kinds of transport mechanisms, and some kinds of regulatory mechanisms. In the 1st step, the authors confirmed that E. coli contains a very high proportion of paralogous **proteins.** Next, the authors defined 2 main classes of paralogous **proteins.** One class is formed of **proteins** which contain a unique structural segment homologous to a single set of related **proteins** . The other class corresponds to **proteins** which contain >1 structural segment of homol., each segment homologous to unrelated sets of **proteins.** Such an independent structural segment of homol. is defined as a module. This modular structure (mean length equivalent to 209 amino acids) corresponds often to entire **proteins**, but there are also **proteins** that appear to be assembled from 2 or 3 independent modules having independent origins. Most multimodular **proteins** appear to have been formed early in their history; a minority appear to be relatively recent **fusions** of independent modules. Examining 1404 independent structural segments of homol., composed of both modules and entire **proteins**, it was found that the segments of **homol. fell** into 352 sequence-related groups or families. The majority of these families (ranging from 2 to 62 members) were functionally homogeneous. This strongly suggests that the 1404 present-day modules and **proteins** derive from a minimal set of 352 ancestral modules, each one being already of the same size and having a function similar to all members of its progeny.

L29 ANSWER 24 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:357083 HCAPLUS
DOCUMENT NUMBER: 127:76474
TITLE: Epidermal growth factor induces **CD44** gene **expression** through a novel regulatory element in mouse fibroblasts
AUTHOR(S): Zhang, Ming; Wang, Ming Hui; Singh, Raj K.; Wells, Alan; Siegal, Gene P.
CORPORATE SOURCE: Departments Pathology, Cell Biology Surgery, University Alabama at Birmingham, Birmingham, AL, 35233-1924, USA
SOURCE: Journal of Biological Chemistry (1997), 272(22), 14139-14146
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Growth factors coordinately regulate a variety of genes associated with pathol. states including tumor invasion and metastasis. Overexpressed epidermal growth factor receptor (EGFR) on tumor cell surfaces is associated with enhanced cell attachment and migration into extracellular matrixes, which promotes tumor aggressiveness. We have demonstrated that epidermal growth factor (EGF) up-regulates the cell surface adhesion mol. **CD44** at both the mRNA and **protein** levels on mouse fibroblasts **expressing** full-length wild-type EGFR (NR6-WT) but not on EGFR-deficient cells (NR6-P). This increases cell attachment to hyaluronic acid. In this investigation, transcriptional regulation of **CD44** by EGF was confirmed by defining an EGF-regulatory element. By employing human **CD44** gene promoter-chloramphenicol acetyltransferase (CAT) constructs transfected into NR6-WT cells, EGF inducibility was observed within a 120-base pair (bp) **DNA** fragment

located 450 bp upstream of the RNA initiation site. Differential EGF inducibility was found among different cell lines chosen, indicating a 3.2- and 1.8-fold enhancement in DU145 cells carrying exogenous wild-type EGFR and in MCF-7 cells, resp., while minimal EGF induction was found in cervical cancer HeLa cells. Utilizing gel shift assays, a time-dependent increase of **DNA-protein** complex formation was found upon EGF stimulation in NR6-WT cells but not in NR6-P cells. Based upon these observations, a novel 22-bp EGF regulatory element (ERE) (5'--604CCCTCTCTCCAGCTCCTCTCCC-583-3') was isolated from the **CD44** gene promoter. This ERE conferred **DNA-protein** binding ability in vitro, as well as the full functional recovery of EGF inducibility of CAT activity when linked to a **homologous** **CD44** promoter or a SV40 promoter driving a CAT reporter gene. A two-base mutation of the ERE completely eliminated its binding activity as well as its EGF inducibility of CAT **expression.** Our studies indicate that EGF induces **CD44** gene **expression** through an interaction between a specific ERE and putative novel transcriptional factor so as to regulate cell attachment to extracellular matrix.

L29 ANSWER 25 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:254239 HCAPLUS
DOCUMENT NUMBER: 126:316286
TITLE: Hyaluronate-CD44 interactions can induce murine B-cell activation
AUTHOR(S): Rafi, Asimah; Nagarkatti, Mitzi; Nagarkatti, Prakash S.
CORPORATE SOURCE: Div. Microbiol. Immunol., Virginia-Maryland Regional Coll. Veterinary Med., Blacksburg, VA, 24061, USA
SOURCE: Blood (1997), 89(8), 2901-2908
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: Saunders
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **CD44** is a widely distributed cell surface glycoprotein whose principal ligand has been identified as hyaluronic acid (HA), a major component of the extracellular matrix (ECM). Recent studies have demonstrated that activation through **CD44** leads to induction of effector function in T cells and macrophages. In the current study, we investigated whether HA or **monoclonal** antibodies (MoAbs) against **CD44** would induce a proliferative response in mouse lymphocytes. Spleen cells from normal and nude, but not severe combined immunodeficient mice, exhibited strong proliferative responsiveness to stimulation with soluble HA or anti-**CD44** MoAbs. Furthermore, purified B cells, but not T cells, were found to respond to HA. HA was unable to stimulate T cells even in the presence of antigen presenting cells (APC) and was unable to act as a costimulus in the presence of mitogenic or submitogenic concns. of anti-CD3 MoAbs. In contrast, stimulation of B cells with HA in vitro, led to B-cell differentiation as measured by production of IgM antibodies in addition to increased **expression** of **CD44** and decreased levels of CD45R. The fact that the B cells were responding directly to HA through its binding to **CD44** and not to any contaminants or endotoxins was demonstrated by the fact that F(ab)2 **fragments** of anti-**CD44** MoAbs or soluble **CD44** **fusion proteins** could significantly inhibit the HA-induced proliferation of B cells. Also, HA-induced proliferation of B cells was not affected by the addition of polymyxin B, and B cells from lipopolysaccharide (LPS)-unresponsive C3H/HeJ strain responded strongly to stimulation with HA. Furthermore, HA, but not chondroitin-sulfate, another major component of the ECM, induced B-cell activation. It was also noted that injection of HA i.p., triggered splenic B cell proliferation in vivo. The current study demonstrates that interaction between HA and **CD44** can regulate murine B-cell effector

functions and that such interactions may play a critical role during normal or autoimmune responsiveness of B cells.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 26 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:185275 HCAPLUS
DOCUMENT NUMBER: 126:263032
TITLE: CD44 is not required for poliovirus replication
AUTHOR(S): Bouchard, Michael J.; Racaniello, Vincent R.
CORPORATE SOURCE: Dep. Microbiology, Columbia Univ. College Physicians Surgeons, New York, NY, 10032, USA
SOURCE: Journal of Virology (1997), 71(4), 2793-2798
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The identification of a **monoclonal** antibody, AF3, which recognizes a single isoform of the cell surface **protein** **CD44** and preferentially blocks binding of serotype 2 poliovirus to HeLa cells, suggested that **CD44** might be an accessory mol. to Pvr, the cell receptor for poliovirus, and that it could play a role in the function of the poliovirus receptor site. We show here that only AF3 blocks binding of serotype 2 poliovirus to HeLa cells and, in contrast to a previously published report, that the anti-**CD44** **monoclonal** antibodies A3D8 and IM7 are unable to block binding to poliovirus. To determine whether **CD44** is involved in poliovirus infection, we analyzed the replication of all three serotypes of poliovirus in human neuroblastoma cells which lack or **express** **CD44** and in mouse neuroblastoma cells which lack Pgp-1, the mouse **homolog** of human **CD44**, and which **express** Pvr. All three poliovirus serotypes replicate with normal kinetics and to normal levels in the absence or presence of **CD44** or in the absence of Pgp-1. Furthermore, the binding affinity consts. of all three poliovirus serotypes for Pvr are unaffected by the presence or absence of **CD44** in the human neuroblastoma cell line. We conclude that **CD44** and Pgp-1 are not required for poliovirus replication and are unlikely to be involved in poliovirus pathogenesis.

L29 ANSWER 27 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:707243 HCAPLUS
DOCUMENT NUMBER: 126:6175
TITLE: Hyaluronan (HA) fragments induce chemokine gene **expression** in alveolar macrophages: the role of HA size and **CD44**
AUTHOR(S): McKee, Charlotte M.; Penno, Margaret B.; Cowman, Mary; Burdick, Marie D.; Strieter, Robert M.; Bao, Clare; Noble, Paul W.
CORPORATE SOURCE: Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, 21205, USA
SOURCE: Journal of Clinical Investigation (1996), 98(10), 2403-2413
CODEN: JCINAO; ISSN: 0021-9738
PUBLISHER: Rockefeller University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Hyaluronan (HA) is a glycosaminoglycan constituent of extracellular matrix. In its native form HA exists as a high mol. weight polymer, but during inflammation lower mol. weight fragments accumulate. The authors identified a collection of inflammatory genes induced in macrophages by HA fragments but not by high mol. weight HA. These include several members of the chemokine gene family: macrophage inflammatory **protein**

-1α, macrophage inflammatory **protein**-1β, cytokine responsive gene-2, monocyte chemoattractant **protein**-1, and regulated on activation, normal T cell **expressed** and secreted (RANTES). HA fragments as small as hexamers are capable of inducing **expression** of these genes in a mouse alveolar macrophage cell line, and **monoclonal** antibody to the HA receptor **CD44** completely blocks binding of fluorescein-labeled HA to these cells and inhibits HA-induced gene **expression.** The authors also investigated the ability of HA fragments to induce chemokine gene **expression** in human alveolar macrophages from patients with idiopathic pulmonary fibrosis and found that interleukin-8 mRNA is markedly induced. Thus, HA fragments generated during inflammation induce the **expression** of macrophage genes which are important in the development and maintenance of the inflammatory response.

L29 ANSWER 28 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:590731 HCAPLUS
DOCUMENT NUMBER: 125:268471
TITLE: The cell adhesion molecule, GP116, is a new CD44 variant (ex14/v10) involved in hyaluronic acid binding and endothelial cell proliferation
AUTHOR(S): Lokeshwar, Vinata B.; Iida, Naoko; Bourguignon, Lilly Y. W.
CORPORATE SOURCE: Dep. Cell Biol. Anat., Univ. Miami Sch. Med., Miami, FL, 33101, USA
SOURCE: Journal of Biological Chemistry (1996), 271(39), 23853-23864
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In this study we have found that endothelial cells from different origins all contain a **CD44**-related transmembrane glycoprotein, named GP116. Using a bovine aortic endothelial cell line and a standard pulse-chase protocol, we show that GP116 is synthesized as a 52-kDa nascent **polypeptide** precursor (p52) which is processed to GP116 as follows, p52 → p63/65 → p82 → p100 → GP116. GP116 contains ≈8 N- and ≈11 O-linked oligosaccharide chains (but lacks glycosaminoglycans) and interacts directly with the cytoskeletal **protein**, ankyrin, both in vitro (Kd ≈1.2 nM) and in vivo. The results of GP116 amino acid composition, reverse transcriptase-polymerase chain reaction, Southern blot, Northern blot, **cloning**, and sequence analyses indicate that endothelial cells **express** this new **CD44** variant that contains an exon having significant **homol.** with human **CD44** exon 14 (ex14/v10). GP116, designated as **CD44** (ex14/v10), has been shown to be a major hyaluronic acid (HA) receptor (Kd ≈0.5-0.8 nM) responsible for cell adhesion. Most importantly, we have found that the interaction between **CD44** (ex14/v10) and HA or a small fragment of HA (10-15 disaccharide units) induces a mitogenic response in endothelial cells. These findings suggest that this **CD44** variant plays an important role in regulating endothelial cell proliferation.

L29 ANSWER 29 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:359752 HCAPLUS
DOCUMENT NUMBER: 125:26304
TITLE: Hyaluronic acid and derivatives for modulation of cellular activity
INVENTOR(S): Asculai, Samuel Simon
PATENT ASSIGNEE(S): Hyal Pharmaceutical Corporation, Can.

SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 23
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9606622	A1	19960307	WO 1995-CA477	19950811
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT				
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2131130	AA	19960301	CA 1994-2131130	19940830
CA 2145605	AA	19960928	CA 1995-2145605	19950327
AU 9531595	A1	19960322	AU 1995-31595	19950811
EP 778776	A1	19970618	EP 1995-927605	19950811
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
HU 76846	A2	19971229	HU 1997-1507	19950811
JP 10504828	T2	19980512	JP 1996-508371	19950811
ZA 9507223	A	19960401	ZA 1995-7223	19950829
CN 1130532	A	19960911	CN 1995-116995	19950829
AU 9672721	A1	19980515	AU 1996-72721	19961018
AU 739701	B2	20011018		
EP 952855	A1	19991103	EP 1996-934250	19961018
R: DE, FR, GB, IT, SE				
NZ 335259	A	20001222	NZ 1996-335259	19961018
ZA 9608847	A	19970527	ZA 1996-8847	19961022
US 6475795	B1	20021105	US 1997-860696	19970616
US 2003036525	A1	20030220	US 2002-234355	20020904

PRIORITY APPLN. INFO.:

CA 1994-2131130	A	19940830
CA 1995-2145605	A	19950327
US 1995-468328	A2	19950606
WO 1995-CA477	W	19950811
WO 1996-CA700	W	19961018
US 1997-860696	A1	19970616

AB A method is provided for the modulation of cellular activity of tissue and cells **expressing** a high affinity cell-surface receptor for the hyaluronic acid, e.g. an adhesion mol. (e.g., ICAM-1, HARLEC, **CD44**) and a regulatory mol. (e.g., RHAMM) of a human. The method comprises the administration of a non-toxic effective amount of a form of hyaluronic acid [e.g., hyaluronic acid, a salt thereof, (e.g., sodium hyaluronate having a mol. weight of less than 750,000 daltons, (e.g., 225,000 daltons)), e.g. from Hyal Pharmaceutical Corp. within the range of 150,000-225,000 daltons and those disclosed in U. S. Patent Application 08/143,983, mol. weight fractions of a form of sodium hyaluronate (e.g., fractions disclosed in Canadian Letters Patent 1205031 (to Fidia)) such as those from 50,000-100,000 daltons, 250,000-350,000 daltons, and 500,000-730,000 daltons, or other fractions, homologues, analogs, derivs., complexes, esters, fragments, and/or subunits of hyaluronic acid and/or combinations thereof] and/or hyaluronic acid-mimicking mols. to a human to modulate cellular activity of tissues and/or cells **expressing** a high affinity cell-surface receptor for hyaluronic acid, e.g., an adhesion mol. and a regulatory mol. in the human body, in a pharmaceutical excipient tolerable by the human (e.g., sterile water). Dosage amts. of pharmaceutical compns. are also disclosed. The methodol. of the invention is useful for the treatment of e.g. cold, stroke, inflammatory process, fibrosis, or cancer. Studies were performed to determine if accessible

hyaluronic acid binding sites are present in tumor tissue in vivo, and the relation of these possible sites to previously described hyaluronic acid-binding **proteins.** Also, further evidence is presented that HARLEC/ICAM-1 is a receptor for hyaluronic acid, that hyaluronic acid also targets human tumors in nude rats, and that the targeting is mainly via binding to HARLEC/ICAM-1 on tumor endothelium.

L29 ANSWER 30 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:118472 HCAPLUS
DOCUMENT NUMBER: 124:199223
TITLE: Oncogene-dependent **expression** of **CD44** in Balb/c 3T3 **derivatives**: correlation with metastatic competence
AUTHOR(S): Kogerman, Priit; Sy, Man-Sun; Culp, Lloyd A.
CORPORATE SOURCE: School of Medicine, Case Western Reserve University, Cleveland, OH, 44106, USA
SOURCE: Clinical & Experimental Metastasis (1996), 14(1), 73-82
CODEN: CEXMD2; ISSN: 0262-0898
PUBLISHER: Rapid Science Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Oncogene-dependent regulation and tumor relatedness of **CD44** **expression** were investigated in Balb/c 3T3 cells and their derivs. transformed with different ras oncogenes (metastatic tumor model) or the human c-sis oncogene (non-metastatic model). Ras transformants using either the Harvey or Kirsten oncogenes **expressed** high levels of cell surface **CD44** **protein** that bound fluoresceinated hyaluronan (HA). Much lower levels of **CD44** were **expressed** in parental 3T3 cells, ras- revertants generated from Kirsten-transformed cells, or c-sis transformants, confirming the significance of the ras oncogene in this upregulation. To determine whether endogenous HA regulates these parameters, hyaluronidase treatment of ras transformants exposed more cell surface **CD44** to anti-**CD44** antibody and increased fluoresceinated HA binding; this did not occur with 3T3 or c-sis transformants. **CD44** **expression** and its HA-binding function were conserved in a panel of in vivo primary and lung metastatic tumor cell lines derived from ras transformants. Ras transformants also retained the ability to down-regulate **CD44** **protein** levels in confluent cultures which occurred through a translational or post-translational mechanism (as **CD44** mRNA levels were not reduced). These results taken together demonstrate that ras-dependent regulation of **CD44** may correlate with tumor progression and metastasis in vivo, possibly (although not exclusively) supporting **CD44**'s importance in metastatic progression.

L29 ANSWER 31 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:98197 HCAPLUS
DOCUMENT NUMBER: 124:143551
TITLE: In vitro culture of human peripheral blood monocytes induces hyaluronan binding and up-regulates monocyte variant **CD44** isoform **expression**
AUTHOR(S): Levesque, Marc C.; Haynes, Barton F.
CORPORATE SOURCE: Dep. Medicine, Duke University Medical Center, Durham, NC, 27710, USA
SOURCE: Journal of Immunology (1996), 156(4), 1557-65
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **CD44** is a cell surface proteoglycan **homologous** to

cartilage link **protein** that serves as a receptor for hyaluronan (HA). **CD44** isoforms include an unspliced 80- to 90-kDa standard form (CD44S) and isoforms derived from alternative splicing of nine **CD44** variant exons (CD44V). Ligation of **CD44** isoforms on monocytes induces the production of IL-1 and TNF-α. In addition, **CD44** mAbs and HA inhibit HIV infection of monocytes by monocytotropic HIV, but do not inhibit T cell tropic HIV infectivity of T cells. To determine the ability of PB lymphocytes and monocytes to bind HA and to define and compare **CD44** isoforms present on PB monocytes and lymphocytes, we studied PBMC using a panel of **CD44** mAbs, HA-FITC, flow cytometry, and Western blot anal. We found that freshly isolated PB monocytes and lymphocytes did not bind soluble HA. However, in vitro culture of PBMC for 8 to 16 h resulted in **CD44**-dependent HA-FITC binding to monocytes, but not to lymphocytes. Western blot and flow cytometry analyses using **CD44** mAbs demonstrated selective **expression** of high m.w. CD44V isoforms on cultured monocytes, but not on lymphocytes. Finally, tissue macrophages and multinucleated giant cells from patients with inflammatory lesions **expressed** CD44V6- and CD44V9-containing **CD44** isoforms in vivo, suggesting that CD44V **expression** is associated with differentiation of monocytes to tissue macrophages in vivo in inflammatory sites. Our data demonstrate that PB monocytes, but not T or B lymphocytes, acquire the ability to bind HA and up-regulate CD44V **expression** after in vitro culture.

L29 ANSWER 32 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:18730 HCAPLUS
DOCUMENT NUMBER: 124:77776
TITLE: The cloning and expression of CD44H extracellular domain cDNA in E. coli
AUTHOR(S): Luo, Zhenge; Gao, Jieying; Liu, Xuebo; Kong, Xiangying; Zhu, Xihua
CORPORATE SOURCE: Inst. Microbiol. Epidemiol., Acad. Milit. Sci., Beijing, 200850, Peop. Rep. China
SOURCE: Zhongguo Mianyixue Zazhi (1995), 11(5), 262-5
CODEN: ZMZAEE; ISSN: 1000-484X
PUBLISHER: Zhongguo Mianyixue Zazhi Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB A cDNA **fragment** of type H antigen **CD44** was amplified by PCR in vitro by using plasmid pUC/**CD44** as temple. The PCR products were digested by EcoRI and BclI, then the obtained signal **peptides** CD44H and CD44H transcellular domain cDNA fragments were inserted into **fusion protein expressive vector** PEX31b. The **recombinant** plasmid PEX-**CD44** was introduced into E. coli RR1 (PCI 857). After induction, a high level **expression** of MS2-**CD44 fusion protein** in E. coli was observed The product might partially purified to 85% purity by simple inclusion body centrifugation. ELISA and western-blot results indicated that the **expressive** MS2-**CD44 protein** could be recognized by anti-CD4 McAb with strong specificity.

L29 ANSWER 33 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:16970 HCAPLUS
DOCUMENT NUMBER: 124:114924
TITLE: Characterization of an anti-CD44 single-chain FV antibody that stimulates natural killer cell activity and induces TNFα release
AUTHOR(S): Tan, Philip H.; Sandmaier, Brenda M.; Stayton, Patrick S.
CORPORATE SOURCE: Center Bioengineering, University Washington, Seattle, WA, 98195, USA
SOURCE: Immunological Investigations (1995), 24(6), 907-26

CODEN: IMINEJ; ISSN: 0882-0139
PUBLISHER: Dekker
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We report the functional characterization of a single-chain Fv (scFv) constructed from an anti-**CD44** mAb (S5) that abrogates marrow rejection in a mismatched canine donor transplant model. The variable light chain (VL) and variable heavy chain (VH) domains of the parent anti-**CD44** antibody were **cloned** and exact match PCR primers designed that spliced the mature variable domains together through a 15 amino acid [Gly4Ser]3 linker-encoding sequence. This gene was put under the control of a T7 promoter and **expressed** in Escherichia coli in insol. inclusion bodies. The scFv was refolded in a cystine/cysteine redox buffer and purified to homogeneity using anion exchange chromatog. The concentration-dependent binding isotherm of the S5 scFv was determined using both direct binding and competitive inhibition flow cytometry assays. S5 scFv effectively blocked FITC-conjugated MAb S5 binding to canine peripheral blood mononuclear cells (PBMC), possessing a mean EC50 (15 nM) equivalent to Fab' fragments of parental S5 (14.7 nM) and approx. two-fold higher than Mab S5 (6 nM). It also binds directly to canine PBMC and possesses a mean EC50 similar to that of the Fab' fragments (1.01 nM vs 1.03 nM). The **recombinant** S5 scFv also retains the potent biol. activity of the parent Mab, stimulating the activation of natural killer (NK) cell activity and the release of tumor necrosis factor alpha (TNFα) in canine PBMC. Like the parent antibody, scFv crossreacted with human **CD44** as examined by direct binding to human PBMC in the flow cytometry assay as well as direct binding to human **CD44** Ig **fusion protein** in an ELISA. It was also able to induce TNFα release in human PBMC. These results support previous work suggesting that monovalent binding is sufficient to generate the in vitro biol. activity of S5. The scFv S5 antibody will thus serve as a useful model for elucidating the mechanism of antibody abrogated marrow rejection and may serve as a human therapeutic agent.

L29 ANSWER 34 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:950261 HCAPLUS
DOCUMENT NUMBER: 123:337417
TITLE: Increased **expression** of **CD44** in bovine articular chondrocytes by catabolic cellular mediators
AUTHOR(S): Chow, Geraldine; Knudson, Cheryl B.; Homandberg, Gene; Knudson, Warren
CORPORATE SOURCE: Dep. Biochem. Pathol., Rush-Presbyterian-St. Luke's Med. Cent., Chicago, IL, 60612, USA
SOURCE: Journal of Biological Chemistry (1995), 270(46), 27734-41
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Bovine articular chondrocytes cultured in alginate beads were used to study the effect of catabolic cellular mediators on **CD44** **expression.** Treatment with either the 29-kDa fragment of fibronectin or interleukin-1α results in a time- and dose-dependent inhibition of proteoglycan synthesis as well as a stimulation in the **expression** of **CD44** mRNA level as determined by semiquant. polymerase chain reaction following reverse transcription. No noticeable effect at 6 h was observed By 24 h, the major **CD44** product (CD44H) from fibronectin **fragment**-treated cultures showed an 8-fold increase; CD44H from interleukin-1α-treated cultures showed a 6-fold increase as compared to control cultures. In addition, a minor band, determined

to be an isoform of **CD44**, was also shown to be up-regulated by both mediators. Stimulation of **CD44** mRNA via interleukin-1 was also evident by in situ hybridization studies of bovine as well as human articular cartilage in organ culture. The increase in **CD44** mRNA is matched by an increase at the **protein** level as determined by Western blot anal. The Western blot reveals a doublet **protein** band at 80-90 kDa that corresponds to the mol. mass of CD44H. Cultures incubated with fibronectin fragments for 24 h had an 8.0-fold increase in **CD44**, while a 6.6-fold was observed for interleukin-1α. Fluorescein-conjugated hyaluronan binding and internalization studies indicate that the increase in **CD44 protein**, induced by interleukin-1α, closely correlates with an increase in functional hyaluronan receptors present at the chondrocyte cell surface. Thus, conditions that up-regulate chondrocyte catabolism also up-regulate the **expression** of **CD44**, a cell surface hyaluronan receptor involved in hyaluronan endocytosis.

L29 ANSWER 35 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:911278 HCAPLUS
DOCUMENT NUMBER: 124:26858
TITLE: Human mammary carcinomas **express** homologs of rat metastasis-associated variants of **CD44**
AUTHOR(S): Sinn, Hans-Peter; Heider, Karl-Heinz; Skroch-Angel, Petra; von Minckwitz, Gunter; Kaufmann, Manfred; Herrlich, Peter; Ponta, Helmut
CORPORATE SOURCE: Department Pathology, University Heidelberg, Heidelberg, D-69120, Germany
SOURCE: Breast Cancer Research and Treatment (1995), 36(3), 307-13
CODEN: BCTRD6; ISSN: 0167-6806
PUBLISHER: Kluwer
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Splice variants of **CD44 expressed** in a metastasizing cell line derived from a rat pancreatic adenocarcinoma have been shown recently to confer metastatic potential onto non-metastasizing rat pancreatic carcinoma and sarcoma cell lines. Homologus of these variants have also been detected in a variety of human malignancies. Using antibodies raised against a bacterially **expressed fusion protein** containing variant **CD44** sequences, we have explored the **expression** of variant **CD44** glycoproteins on tumors of the female breast. The material examined included normal tissue, hyperplastic lesions, 103 primary invasive mammary carcinomas, 10 in situ carcinomas, 12 local recurrences and 18 lymph node metastases. Using a **polyclonal** serum directed against several variant **CD44** epitopes, normal mammary epithelia as well as ductal hyperplasias were neg. for these splice variants, while the variant **CD44** epitopes were detectable in all but six of the primary invasive carcinomas. From the reaction with various **monoclonal** antibodies and **polyclonal** sera specific for individual epitopes it is obvious that the tumors predominantly **express CD44** variants encoded by exons v5 to v7. Interestingly, all investigated lymph node metastases reacted pos. with the variant-specific antibodies, in contrast to primary tumors which reacted in 54% to 86% of the cases, depending on the antibody used. Statistical anal. revealed a significant correlation between **expression** of variant exons v3/v4 and v6 and increased tumor grade ($p = 0.001$ and $p < 0.05$, resp.; Fisher's exact test). Exon v6 is carried by the variants which confer metastatic capability in the rat. These results indicate that the **expression** of the **CD44** variants is upregulated in mammary carcinomas and is closely linked to tumor anaplasia.

L29 ANSWER 36 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:827311 HCAPLUS
DOCUMENT NUMBER: 123:225901
TITLE: CD44 (Pgp-1) inhibits CD3 and dexamethasone-induced apoptosis
AUTHOR(S): Ayroldi, E.; Cannarile, L.; Migliorati, G.; Bartoli, A.; Nicoletti, I.; Riccardi, C.
CORPORATE SOURCE: Department of Clinical Medicine, Perugia University Medical School, Perugia, Italy
SOURCE: Blood (1995), 86(7), 2672-8
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: Saunders
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Anti-CD3 **monoclonal** antibodies (MoAbs) and glucocorticoid hormones (GCH) induce apoptosis in immature thymocytes and peripheral T lymphocytes. This process is inhibited by a number of growth factors, including interleukin-2 (IL-2), IL-3, and IL-4, indicating that signals generated by membrane receptors can modulate the survival of lymphoid cells. To investigate whether signals activated by adhesion receptors have a similar activity, the authors analyzed the effect of **CD44** (Pgp-1) adhesion mol. receptor stimulation on T-cell apoptosis induced by 3 stimuli [anti-CD3 MoAbs, dexamethasone (DEX) treatment, and exposure to UV irradiation] on a 3DO T-cell line. The results show that **CD44** engagement, either by hyaluronic acid (HA) or anti-**CD44** MoAbs, inhibits **DNA fragmentation** and apoptosis induced by DEX and anti-CD3 MoAbs, whereas that induced by UV, a p53-dependent phenomenon, was not inhibited. Furthermore, the anti-apoptotic effect exerted through **CD44** activation does not seem related to overexpression of bcl-2 or to have appreciable effects on cell proliferation. Thus, adhesion mols. modulate T-cell survival by counteracting apoptosis induced by DEX or anti-CD3 MoAbs.

L29 ANSWER 37 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:723814 HCAPLUS
DOCUMENT NUMBER: 123:160602
TITLE: Quantitative immunocytochemical study of secretory protein expression in parotid glands of rats chronically treated with isoproterenol
AUTHOR(S): Vugman, Ithamar; Hand, Arthur R.
CORPORATE SOURCE: Clinical Investigations and Patient Care Branch, National Institute of Dental Research, Bethesda, MD, 20892, USA
SOURCE: Microscopy Research and Technique (1995), 31(2), 106-17
CODEN: MRTEEO; ISSN: 1059-910X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Chronic treatment of mice and rats with isoproterenol (IPR) causes marked hypertrophy and hyperplasia of the salivary glands, and alters the **expression** of several secretory **proteins.** The authors used quant. postembedding immunogold labeling to study the cellular responses in the rat parotid gland during daily (≤10 days) injections of IPR and during recovery (≤14 days) after cessation of IPR treatment. Labeling densities of acinar cell secretory granules with antibodies to amylase and **protein** SMG-B1 (cross-reactive with the rat **homolog** of Parotid Secretory **Protein**, PSP) **fell** to 10% of control levels after 8-10 IPR injections, then increased during recovery, paralleling previous biochem. detns. of changes in **protein** and mRNA levels. With antibodies to proline-rich **proteins** (PRP), labeling densities initially **fell**, then subsequently showed considerable variability, but never exceeded control

levels. These results contrast with biochem. detns. showing a marked induction of PRP synthesis, and may have both immunol. and structural explanations. Occasional intercalated duct cells located close to the acini underwent differentiation toward an acinar-like phenotype as a result of IPR treatment. After 1-2 IPR injections, the secretory granules of these cells were labeled with antibodies to amylase and PRP. Subsequently, the granules appeared to be electron-lucent and were increased in size and number These observations support earlier work, suggesting that intercalated duct cells may differentiate into other gland cell types.

L29 ANSWER 38 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:688235 HCAPLUS
DOCUMENT NUMBER: 123:122895
TITLE: Pharmacokinetics of [3H]biotin bound to different avidin analogs
AUTHOR(S): Kang, Young-Sook; Saito, Yasunari; Pardrigge, William M.
CORPORATE SOURCE: Dep. Medicine, UCLA School Medicine, Los Angeles, CA, 90024-1682, USA
SOURCE: Journal of Drug Targeting (1995), 3(2), 159-65
CODEN: JDTAEH; ISSN: 1061-186X
PUBLISHER: Harwood
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The use of avidin-biotin technol. in drug delivery facilitates the conjugation of biotinylated therapeutics to transport **vectors** that are enabled to undergo receptor-mediated transcytosis through the brain capillary endothelial wall, which makes up the blood-brain barrier (BBB) in vivo. However, the conjugation of avidin, a cationic glycosylated **protein**, to transport **vectors** greatly increases the rate of removal of the **vector** from the blood stream, owing to rapid uptake of avidin by peripheral tissues such as liver and kidney. However, modified avidins may retain high affinity biotin binding properties, but may not be rapidly removed from plasma by peripheral tissues, and such avidin analogs would provide preferred plasma pharmacokinetic profiles. Therefore, the present studies investigate the pharmacokinetics of plasma removal of [3H]biotin bound to one of six different avidin analogs: streptavidin, Neutra-lite avidin, avidin, neutral avidin, Lite-avidin, and succinylated avidin. Isoelec. focusing studies show that avidin and Lite-avidin were highly cationic **proteins**, whereas neutral avidin, Neutra-lite avidin, and streptavidin were neutral **proteins**, and succinylated avidin had an acidic isoelec. point. The avidin **analogs fell** into two groups with respect to rate of biotin removal from plasma. The low clearance group included streptavidin and Neutra-lite avidin, which had a mean plasma clearance of 0.41 mL/min/kg. The high clearance group consisted of succinylated avidin, neutral avidin, and Lite-avidins. In conclusion, these studies show that the rate of removal of avidin analogs differs by more than a log order of magnitude depending on the charge and the degree of glycosylation of the avidin analog. Use of high clearance avidin analogs may be preferred when it is desired to rapidly remove biotinylated therapeutics from the plasma, whereas the use of low clearance avidins may be desired in targeted drug delivery.

L29 ANSWER 39 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:665896 HCAPLUS
DOCUMENT NUMBER: 123:249768
TITLE: Differential expression of peroxidase isogenes during the early stages of infection of the tropical forage legume Stylosanthes humilis by Colletotrichum gloeosporioides

AUTHOR(S): Harrison, Stuart J.; Curtis, Mark D.; McIntyre, C. Lynne; Maclean, Donald J.; Manners, John M.
CORPORATE SOURCE: Cooperative Research Centre Tropical Plant Pathology, University Queensland, Brisbane, 4072, Australia
SOURCE: Molecular Plant-Microbe Interactions (1995), 8(3), 398-406
CODEN: MPMIEL; ISSN: 0894-0282
PUBLISHER: American Phytopathological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Infection of Stylosanthes humilis by the fungal phytopathogen Colletotrichum gloeosporioides is associated with an increase in peroxidase enzyme activity within 24 h postinoculation. Peroxidase gene **expression** was investigated as a first step towards understanding the regulation and functional importance of this **host** response to fungal attack. Four distinct cDNAs Shpx 2, 5, 6, and 12, isolated from a cDNA library of S. humilis contained deduced amino acid (aa) sequence motifs characteristic of peroxidases. Three of these (Shpx 2, 5, and 6) were full-length and their deduced **proteins** each **fell** into a different **homol.** group based on comparisons with other plant peroxidases. Each cDNA appeared to hybridize to only one or two genes in S. humilis. MRNAs corresponding to Shpx2, Shpx6, and Shpx12 were **expressed** relatively abundantly in young leaves, with lesser **expression** of Shpx2 and Shpx6 and no **expression** of Shpx12 detected in roots. No **expression** of these genes was detected in stems or old leaves. The mRNA of Shpx5 was relatively abundant in stems and to a lesser extent in young leaves with C. gloeosporioides greatly increased **expression** of the mRNAs of Shpx2 and Shpx6 but not Shpx5 nor Shpx12 compared to mock-inoculated controls. The mRNA of Shpx6 was strongly induced by the pathogen 4 h postinoculation, a time which precedes fungal penetration, while Shpx2 was induced to higher levels than controls at 24 h after inoculation. The mRNAs of both Shpx2 and Shpx6 but not Shpx5 and Shpx12 were also induced by wounding. These results indicate that specific **host** peroxidase isogenes are induced at very early stages of the interaction of C. gloeosporioides with S. humilis and that **host** recognition of the pathogen appears to occur prior to phys. penetration of the epidermal cell wall.

L29 ANSWER 40 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:596818 HCAPLUS
DOCUMENT NUMBER: 121:196818
TITLE: Biological effects of prostate specific antigen as an insulin-like growth factor binding protein-3 protease
AUTHOR(S): Cohen, P.; Peehl, D. M.; Graves, H. C. B.; Rosenfield, R. G.
CORPORATE SOURCE: Department of Pediatrics, University of Pennsylvania, Philadelphia, PA, 19103, USA
SOURCE: Journal of Endocrinology (1994), 142(3), 407-15
CODEN: JOENAK; ISSN: 0022-0795
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Prostate specific antigen (PSA) is an insulin-like growth factor (IGF) binding **protein**-3 (IGFBP-3) protease found in seminal plasma and produced by prostatic epithelial cells (PC-E) in vivo. The authors examined the effects of PSA-proteolysis of IGFBP-3 on the affinity of IGFBP-3 fragments for IGFs and on the mitogenic action of IGFs on PCE-E. **Recombinant** human IGFBP-3 was cleaved by PSA, then incubated with 125I-IGF-I or -II in the presence of varying concns. of unlabeled **peptides**, and then crosslinking electrophoresis and densitometric anal. were performed. While the affinity of IGF-II for the PSA-generated IGFBP-3 **fragments fell** slightly compared to intact

IGFBP-3, the affinity of the PSA-generated IGFBP-3 **fragments** for IGF-I **fell** by ten fold. The addition of IGF-I or -II to PC-E in serum-free culture conditions resulted in a two-fold stimulation of cell number compared to control. The presence of IGFBP-3 in the media blocked the IGF-induced stimulation, but had no independent effect in the absence of IGFs. When PSA was added to PC-E cultures to which both IGF-I or -II and IGFBP-3 were added, the inhibitory effects of IGFBP-3 on IGF mitogenesis were reversed. Apparently, PSA decreases the affinity of IGFBP-3 for IGF and can potentiate IGF action in the presence of inhibitory IGFBP-3. This phenomenon may contribute to normal and malignant prostate growth.

L29 ANSWER 41 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:555742 HCAPLUS
DOCUMENT NUMBER: 121:155742
TITLE: **Peptides** derived from **CD44** antigens and their use in the modulation of cell adhesion
INVENTOR(S): Haynes, Barton F.; Hale, Laura P.; Patton, Karen L.; Telen, Marilyn J.; Liao, Hua Xin
PATENT ASSIGNEE(S): Duke University, USA
SOURCE: PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9409811	A1	19940511	WO 1993-US10412	19931029
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9455435	A1	19940524	AU 1994-55435	19931029

PRIORITY APPLN. INFO.: US 1992-973339 A 19921030
WO 1993-US10412 W 19931029

AB **CD44**-mediated responses of the immune system, e.g. inflammation, are modulated with **peptides** derived from **CD44** antigens. In particular, the **peptides** are used to suppress T cell activation, inhibit **CD44**-mediated cell adhesion and **CD44**-monocyte IL1 release, as inflammation inhibitors, and in transporting a drug to a site of inflammation. Studies on **CD44** antigen levels in synovial fluid in osteoarthritis and rheumatoid arthritis indicated a role for the antigen in inflammation. Antigenic **peptides** derived from **CD44** were shown to inhibit T cell receptor-mediated activation of T cells.

IT **157147-26-7**, **CD44** antigen **fragment** (human)
157147-27-8, **CD44** antigen **fragment** (human)
157147-28-9, **CD44** antigen **fragment** (human)
157147-29-0, **CD44** antigen **fragment** (human)
157147-30-3, **CD44** antigen **fragment** (human)
157147-31-4, **CD44** antigen **fragment** (human)
157147-32-5, **CD44** antigen **fragment** (human)
157147-33-6, **CD44** antigen **fragment** (human)
157147-34-7, **CD44** antigen **fragment** (human)
157147-35-8, **CD44** antigen **fragment** (human)
157147-36-9, **CD44** antigen **fragment** (human)
157147-37-0, **CD44** antigen **fragment** (human)
157147-38-1, **CD44** antigen **fragment** (human)
157147-39-2, **CD44** antigen **fragment** (human)
157147-40-5, **CD44** antigen **fragment** (human)
157147-41-6, **CD44** antigen **fragment** (human)
157147-42-7, **CD44** antigen **fragment** (human)

157147-43-8, **CD44** antigen **fragment** (human)
157147-44-9, **CD44** antigen **fragment** (human)
157153-40-7, **CD44** antigen **fragment** (human)
157172-83-3, **CD44** antigen **fragment** (human)
157242-80-3, **CD44** antigen **fragment** (human)
157242-82-5, **CD44** antigen **fragment** (human)
157242-88-1, **CD44** antigen **fragment** (human)
157242-93-8, **CD44** antigen **fragment** (human)
157382-35-9, **CD44** antigen **fragment** (human)
RL: BIOL (Biological study)
(as inhibitor of **CD44** action)

L29 ANSWER 42 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:530775 HCAPLUS
DOCUMENT NUMBER: 121:130775
TITLE: ERM family members as molecular linkers between the cell surface glycoprotein CD44 and actin-based cytoskeletons
AUTHOR(S): Tsukita, Sachiko; Oishi, Kumiko; Sato, Naruki; Sagara, Junji; Kawai, Akihiko; Tsukita, Shoichiro
CORPORATE SOURCE: Dep. Information Physiology, Natl. Inst. Physiological Sci., Okazaki, 444, Japan
SOURCE: Journal of Cell Biology (1994), 126(2), 391-401
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The ERM family members, ezrin, radixin, and moesin, localizing just beneath the plasma membranes, are thought to be involved in the actin filament/plasma membrane association To identify the integral membrane **protein** directly associated with ERM family members, the authors performed immunopptn. studies using antimoesin mAb and cultured baby hamster kidney (BHK) cells metabolically labeled with [35S]methionine or surface-labeled with biotin. The results indicated that moesin is directly associated with a 140-kD integral membrane **protein.** Using BHK cells as antigens, the authors obtained a mAb that recognized the 140-kD membrane **protein.** The authors next **cloned** a cDNA encoding the 140-kD membrane **protein** and identified it as **CD44**, a broadly distributed cell surface glycoprotein. Immunopptn. with various anti-**CD44** mAbs showed that ezrin and radixin, as well as moesin, are associated with **CD44**, not only in BHK cells, but also in mouse L fibroblasts. Furthermore, immunofluorescence microscopy revealed that in both BHK and L cells, the Triton X-100-insol. **CD44** is precisely colocalized with ERM family members. The authors concluded that ERM family members work as mol. linkers between the cytoplasmic domain of **CD44** and actin-based cytoskeletons.
IT **157092-27-8**, Glycoprotein **CD44** (hamster **clone** B10 v9/v10-containing fragment)
RL: PRP (Properties)
(amino acid sequence of)

L29 ANSWER 43 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:480741 HCAPLUS
DOCUMENT NUMBER: 121:80741
TITLE: Molecular **cloning** of the canine **CD44** antigen cDNA
AUTHOR(S): Milde, Kerstin F.; Alejandro, Rodolfo; Mintz, Daniel H.; Pastori, Ricardo L.
CORPORATE SOURCE: Diabetes Research Institute, University of Miami School of Medicine, P.O. Box 016960, Miami, FL, 33101, USA
SOURCE: Biochimica et Biophysica Acta (1994), 1218(1), 112-14

CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mol. **cloning** of the dog **homolog** of the human **CD44** was achieved using RT/PCR. A 1055 bp cDNA has a deduced amino acid sequence of 351 residues, 338 of them correspond to the mature **protein.** Nine conserved cysteine residues were found. The extracellular region contains a single link superfamily domain on the N-terminal part and potential post-translational modification sites as: N- and O-linked glycosylation sites and chondroitin sulfate attachment sites. Three mRNAs of 2.2, 3.8 and 4.4 kb were identified on Northern blot anal. and Western blot hybridization revealed a 85-90 kDa **protein** **expressed** in lymph node tissue.
IT **156559-59-0**
RL: BIOL (Biological study)
(amino acid sequence of and **cloning** of gene for)

L29 ANSWER 44 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:267238 HCAPLUS
DOCUMENT NUMBER: 120:267238
TITLE: A novel form of congenital dyserythropoietic anemia associated with deficiency of erythroid CD44 and a unique blood group phenotype [In(a - b -), Co(a - b -)]
AUTHOR(S): Parsons, Stephen F.; Jones, Jeff; Anstee, David J.; Judson, Philip A.; Gardner, Brigitte; Wiener, Edith; Poole, Joyce; Illum, Niels; Wickramasinghe, Sunitha N.
CORPORATE SOURCE: Int. Blood Group Reference Lab., Bristol, UK
SOURCE: Blood (1994), 83(3), 860-8
CODEN: BLOOAW; ISSN: 0006-4971
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have used a panel of well-characterized **monoclonal** antibodies (MoAbs) to examine the blood cells of a patient with a novel form of congenital dyserythropoietic anemia (CDA) characterized by intraerythroblastic and intraerythrocytic membranous inclusions. Twelve antibodies defining three nonoverlapping epitope groups on the extracellular domain of **CD44** all failed to react with the red blood cells (RBCs) of the patient. A rabbit antibody to the cytoplasmic domain of **CD44** from normal RBCs failed to react with the patient's RBC ghosts. In contrast, the patient's lymphocytes, granulocytes, and monocytes showed apparently normal **CD44** **expression.** Bone marrow prepns. stained with **CD44** antibodies and visualized with 15I antimouse Ig (F(ab')2) follows by autoradiog. showed pos. staining of lymphocytes and myeloid cells but not of most orthotolidine-pos. erythroblasts. The patients RBCS also gave weaker than normal reactions with MoAbs of anti-LWab specificity while MoAbs to glycophorins A, B, and C, Rh **polypeptides**, CD47, CD55, CD58, CD59, acetylcholinesterase, and Lutheran and Kell glycoproteins all gave normal reactions. Agglutination tests with human blood grouping sera demonstrated that the RBCs of the patient have the unique phenotype In(a - b -), Co(a - b -) and that they also lack the high incidence RBC antigen AnWj. The phenotype In(a - b -) would be expected because these antigens are known to be **expressed** on **CD44.** There is also some evidence associating the AnWj antigen with **CD44.** However, the CO blood group locus is on chromosome 7p whereas that for **CD44** is on chromosome 11p. Quant. binding assays using 125I-labeled Fab **fragments** of **CD44** antibodies did not show any evidence for reduced levels of CE44 on RBCs from the parents of the patient or from her unaffected sister. The parents and sister had the common Colton blood group phenotype [Co(a + b -)]. Neither deficiency of **CD44** nor absence of Colton antigens are general features of CDA because

erythrocytes from patients with CDA I, CDA II, CDA III, and two other unclassified CDAs had normal **expression** of **CD44** and normal Colton blood group phenotypes. Further anal. of the defect(s) present in the patient's erythroid cells may provide useful information regarding membrane assembly and the regulation of differentiation in normal erythroid cells.

L29 ANSWER 45 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:470365 HCAPLUS
DOCUMENT NUMBER: 119:70365
TITLE: Use of anti-CD44 variant antibody-containing preparations for immunosuppression
INVENTOR(S): Zoeller, Margot; Herrlich, Peter; Ponta, Helmut
PATENT ASSIGNEE(S): Kernforschungszentrum Karlsruhe GmbH, Germany; Deutsches Krebsforschungszentrum Heidelberg; Boehringer Ingelheim International G.m.b.H.
SOURCE: Eur. Pat. Appl., 19 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 538754	A2	19930428	EP 1992-117775	19921017
EP 538754	A3	19940525		
EP 538754	B1	19980114		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
DE 4134982	A1	19930429	DE 1991-4134982	19911023
AT 162079	E	19980115	AT 1992-117775	19921017
ES 2111031	T3	19980301	ES 1992-117775	19921017
CA 2081150	AA	19930424	CA 1992-2081150	19921022
CA 2081150	C	20010410		
NO 9204103	A	19930426	NO 1992-4103	19921022
AU 9227244	A1	19930429	AU 1992-27244	19921022
AU 659687	B2	19950525		
ZA 9208162	A	19930505	ZA 1992-8162	19921022
HU 63063	A2	19930728	HU 1992-3337	19921022
HU 216020	B	19990428		
JP 05310596	A2	19931122	JP 1992-307913	19921023
US 5951982	A	19990914	US 1994-359850	19941220

PRIORITY APPLN. INFO.: DE 1991-4134982 A 19911023
US 1992-963323 B1 19921023

AB (**Monoclonal**) antibodies to tumor cell variants of surface glycoprotein **CD44** (which is responsible for metastasis via the lymphatic system) show immunosuppressant as well as antimetastatic activity, and can be used for treatment, prevention, and diagnosis of immunoregulatory diseases and for treatment of autoimmune, allergic, inflammatory, degenerative, rheumatic, and hyperproliferative diseases and transplant rejection. Preferred **monoclonal** antibodies recognize the epitopic sequence EEAATQKEKW. Thus, cDNA prepared from poly(A)+ RNA from several human cell lines and amplified by PCR contained inserts between nucleotides 782 and 783, of which the longest (1014 bp), from human lung carcinoma cell line LCLC97, comprised 5 domains (exons). **Monoclonal** antibody 1.1ASML against the LCLC97 **CD44** variant inhibited the antigen-induced activation of B-, T-, and cytotoxic T-cells in rats.

IT **148790-21-0**, **CD44** antigen (human large-cell lung carcinoma cell line LCLC97 variant)
RL: PRP (Properties)
(amino acid sequence of and **monoclonal** antibody to, as

immunosuppressant)
IT **140355-90-4**, **Deoxyribonucleic acid** (human cell LCLC97 antigen **CD 44 fragment**-specifying)
148790-22-1, **DNA** (rat tumor cell line BSpASML **CD44** antigen variant cDNA)
RL: PRP (Properties)
(nucleotide sequence of)

L29 ANSWER 46 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:145517 HCAPLUS
DOCUMENT NUMBER: 118:145517
TITLE: Activated human lymphocytes and aggressive non-Hodgkin's lymphomas **express** a homolog of the rat metastasis-associated variant of **CD44**
AUTHOR(S): Koopman, Gerrit; Heider, Karl Heinz; Horst, Eveliene; Adolf, Guenther R.; Van den Berg, Frank; Ponta, Helmut; Herrlich, Peter; Pals, Steven T.
CORPORATE SOURCE: Academic Med. Cent., Univ. Amsterdam, Amsterdam, 1105 AZ, Neth.
SOURCE: Journal of Experimental Medicine (1993), 177(4), 897-904
CODEN: JEMEAV; ISSN: 0022-1007
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A recently described splice variant of **CD44 expressed** in metastasizing cell lines of rat tumors, has been shown to confer metastatic potential to nonmetastasizing rat pancreatic carcinoma and sarcoma cell lines. Using antibodies raised against a bacterial **fusion protein** encoded by variant **CD44** sequences, the authors have explored the **expression** of variant **CD44** glycoproteins on human lymphoid cells and tissues and on non-Hodgkin's lymphomas. Normal lymphohematopoietic cells **express** barely detectable low levels of variant **CD44** glycoproteins, whereas T lymphocytes, upon activation by mitogen or antigen, transiently upregulate **expression** of specific **CD44** variant glycoproteins. The reaction pattern of various antibodies indicates that these **CD44** variants contain the domain encoded by exon v6, which is part of the variant that in the rat confers metastatic capability. It is interesting that overexpression of v6 was also found in several aggressive, but not low-grade, non-Hodgkin's lymphomas.

L29 ANSWER 47 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:58117 HCAPLUS
DOCUMENT NUMBER: 118:58117
TITLE: Cytokine-induced protein TSG-6, DNA coding therefor, and uses thereof
INVENTOR(S): Lee, Tae Ho; Wisniewski, Hans Georg; Vilcek, Jan
PATENT ASSIGNEE(S): New York University, USA
SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9212175	A1	19920723	WO 1992-US333	19920114
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
AU 9212288	A1	19920817	AU 1992-12288	19920114
EP 567575	A1	19931103	EP 1992-904669	19920114

EP 567575 B1 19991013
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE
JP 06504912 T2 19940609 JP 1992-504569 19920114
AT 185573 E 19991015 AT 1992-904669 19920114
PRIORITY APPLN. INFO.: US 1991-642312 A2 19910114
WO 1992-US333 A 19920114

AB Pleiotropic proinflammatory cytokines, such as tumor necrosis factor (TNF) and interleukin-1, induce **expression** of a **protein** mol. termed TSG-6 (TSG = TNF-stimulated gene sequence), in connective tissue cells. The TSG-6 **protein**, and functional derivs. thereof, **DNA** encoding therefor, **expression** vehicles (e.g. a plasmid), and **host** cells transformed with or transfected by the **DNA** mol., as well as methods for producing the **protein** and the **DNA** mol., are provided (nucleotide and corresponding amino acid sequences are included). Antibodies specific for the TSG-6 **protein** are disclosed, as is a method for detecting the presence of TSG-6 in a biol. sample using the antibody or other mol. capable of binding to TSG-6 (e.g. hyaluronic acid). A method for detecting the presence of **nucleic acid** encoding a normal or mutant TSG-6, a method for measuring induction of **expression** of TSG-6 in a cell using either **nucleic acid** hybridization or immunoassay, a method for identifying a compound capable of inducing the **expression** of TSG-6 in a cell, and a method for measuring the ability of a cell to respond to TNF are also provided. A cDNA library was prepared from TNF-treated FS-4 cells, and a variety of TSG cDNAs were isolated. **Homol.** of TSG-6 to **CD44**/Hermes and to the cartilage link **protein** family is described, as are the production of TSG-6-containing **fusion proteins** and the effect of TSG-6 in leukocyte adhesion. TSG-6 was determined in serum and joint fluid samples of patients with a variety of arthritic diseases.

L29 ANSWER 48 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:37260 HCAPLUS
DOCUMENT NUMBER: 118:37260
TITLE: A human homolog of the rat metastasis-associated variant of **CD44** is **expressed** in colorectal carcinomas and adenomatous polyps
AUTHOR(S): Heider, Karl Heinz; Hofmann, Martin; Hors, Eveliene; Van den Berg, Frank; Ponta, Helmut; Herrlich, Peter; Pals, Steven T.
CORPORATE SOURCE: Inst. Genet. Toxikol., Kernforschungszent. Karlsruhe, Karlsruhe, D-7500/1, Germany
SOURCE: Journal of Cell Biology (1993), 120(1), 227-33
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A recently described splice variant of **CD44 expressed** in metastasizing cell lines of rat tumors has been shown to confer metastatic potential to a non-metastasizing rat pancreatic carcinoma cell line and to non-metastasizing sarcoma cells. Homologs of this variant as well as several other **CD44** splice variants are also **expressed** at the RNA level in human carcinoma cell lines from lung, breast, and colon, and in immortalized keratinocytes. Using antibodies raised against a bacterial **fusion protein** encoded by variant **CD44** sequences, the **expression** of variant **CD44** glycoproteins was studied in normal human tissues and in colorectal neoplasia. **Expression** of **CD44** variant **proteins** in normal human tissues was readily found on several epithelial tissues including the squamous epithelia of the epidermis, tonsils, and pharynx, and the glandular epithelium of the pancreatic ducts, but was largely absent from other epithelia and from most non-epithelial cells and tissues. In human colorectal neoplasia

CD44 variant **proteins**, including **homologs** of those which confer metastatic ability to rat tumors, were found on all invasive carcinomas and carcinoma metastases. Interestingly, focal **expression** was also observed in adenomatous polyps, **expression** being related to areas of dysplasia. The distribution of the **CD44** variants in human tissues suggests that they play a role in a few restricted differentiation pathways and that in colorectal tumors one of these pathways has been reactivated. The finding that metastasis-related variants are already **expressed** at a relatively early stage in colorectal carcinogenesis and tumor progression, i.e., in adenomatous polyps, suggests the existence of a yet unknown selective advantage linked to **CD44** variant **expression.** The continued **expression** in metastases would be compatible with a role in the metastatic process.

L29 ANSWER 49 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:424683 HCAPLUS
DOCUMENT NUMBER: 117:24683
TITLE: Surface **protein CD44** variant, cDNA sequence encoding it, antibody to it, and its use in diagnosis and therapy
INVENTOR(S): Herrlich, Peter; Ponta, Helmut; Guenthert, Ursula; Matzku, Siegfried; Wenzel, Achim
PATENT ASSIGNEE(S): Kernforschungszentrum Karlsruhe G.m.b.H., Germany; Universitaet Karlsruhe; Deutsches Krebsforschungszentrum
SOURCE: Ger. Offen., 12 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 4014510	A1	19911114	DE 1990-4014510	19900507
WO 9117248	A1	19911114	WO 1991-EP614	19910330
W: AU, CA, FI, HU, JP, KP, KR, NO, PL, SU, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
AU 9175659	A1	19911127	AU 1991-75659	19910330
AU 646858	B2	19940310		
EP 531300	A1	19930317	EP 1991-906994	19910330
EP 531300	B1	19940608		
R: AT, BE, CH, DE, DK, FR, GB, GR, IT, LI, LU, NL, SE				
HU 63652	A2	19930928	HU 1992-3449	19910330
HU 218905	B	20001228		
JP 05508309	T2	19931125	JP 1991-506825	19910330
JP 3133062	B2	20010205		
AT 106943	E	19940615	AT 1991-906994	19910330
IL 98024	A1	20010614	IL 1991-98024	19910502
ZA 9103389	A	19920325	ZA 1991-3389	19910506
NO 9204234	A	19930104	NO 1992-4234	19921104
NO 2000001668	A	20000331	NO 2000-1668	19921104
US 5506119	A	19960409	US 1992-946497	19921109
US 5760178	A	19980602	US 1995-483322	19950607
US 5885575	A	19990323	US 1995-478882	19950607
FI 9704209	A	19971112	FI 1997-4209	19971112
NO 9800293	A	19930104	NO 1998-293	19980123

PRIORITY APPLN. INFO.:

DE 1990-4014510	A	19900507
EP 1991-906994	A	19910330
WO 1991-EP614	A	19910330
FI 1992-5043	A	19921106

US 1992-946497 A3 19921109

AB A cDNA was isolated which encodes a variant of surface glycoprotein **CD44**, which is involved in lymphocyte adhesion and cell-cell and cell-matrix interaction. The **CD44** variant glycoprotein was isolated from metastasizing rat BSp73ASML tumor cells, and has an extracellular 154-amino-acid insert between residues 220 and 237. **Monoclonal** antibody 1.1ASML to the extracellular domain was used to isolate cDNA encoding the **CD44** variant from a bacterial **expression** library. Hybridization probes prepared from the cDNA were used to show that mRNA coding for the extracellular domain was produced by BSp73ASML cells but not by the nonmetastasizing parent BSp73AS cells. Metastatic potential was correlated with the **expression** of variant **CD44**, and was inhibited by antibody 1.1ASML. The nucleotide sequence for the cDNA and the corresponding derived amino acid sequence for the extracellular domain of the rat **CD44** variant, and **homologous** sequences for the human variant, are presented. Potential applications to diagnosis and therapy include immunohistochem. studies on clin. tumor material, detection of soluble **CD44** variant in the serum, preparation of cytotoxic antibody-toxin conjugates, and injection of the **CD44** variant to block tissue binding sites for metastatic tumor cells.

IT **136896-09-8**, **Deoxyribonucleic acid** (rat **clone** pMeta-1 antigen **CD 44** messenger RNA-complementary) **136896-10-1** **141961-71-9**, **Deoxyribonucleic acid** (human antigen **CD 44** 110-amino acid **fragment**-specifying **fragment**) **141961-72-0**, **Deoxyribonucleic acid** (rat **clone** pMeta-1 203-357-antigen **CD 44**-specifying **fragment**)
RL: PRP (Properties)
(**cloning** and **expression** and nucleotide sequence of, metastasis in relation to)

IT **136894-51-4**, Antigen **CD 44** (rat **clone** pMeta-1 isoform precursor **protein** moiety reduced) **141961-70-8**, 203-357-Antigen **CD 44** (rat **clone** pMeta-1 isoform **protein** moiety reduced)
RL: BIOL (Biological study)
(metastasis-associated, amino acid sequence of)

L29 ANSWER 50 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:149842 HCAPLUS
DOCUMENT NUMBER: 116:149842
TITLE: Multiple variants of the human lymphocyte homing receptor CD44 generated by insertions at a single site in the extracellular domain
AUTHOR(S): Jackson, David G.; Buckley, Jonathan; Bell, John I.
CORPORATE SOURCE: Inst. Mol. Med., John Radcliffe Hosp., Oxford, OX3 9DU, UK
SOURCE: Journal of Biological Chemistry (1992), 267(7), 4732-9
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The human **CD44** cell-surface glycoprotein participates in a wide variety of cell-cell interactions including lymphocyte homing and tumor metastasis. The **CD44** antigen is known to display extensive size heterogeneity when compared between different tissue sources although the structural basis for this variation is not yet clear. The authors obtained evidence for alternative splicing, and report here the **cloning** and sequencing of six different **CD44** sequence variants from a variety of cell lines using a combination of **expression cloning** and the polymerase chain reaction. Comparison of these variants indicates that each is probably assembled by

the insertion of five different exon units in tandem into a discrete site within the membrane proximal region of the extracellular domain. One of the variants contain an exon that shares extensive amino acid sequence **homol.** with a recently described rat **CD44** variant that mediates tumor metastasis. Another variant contains a new exon that encodes a tandem repeat of the consensus sequence SG for covalent modification with chondroitin sulfate and is **expressed** predominantly on mammary tumors. A mechanism of alternative exon splicing is suggested for much of the observed structural heterogeneity of **CD44**. The particular set of **CD44** variants **expressed** in a single cell may represent a precise postal code directing the final destination of migrating cells and metastatic tumors.

L29 ANSWER 51 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:604663 HCAPLUS
DOCUMENT NUMBER: 115:204663
TITLE: A new variant of glycoprotein CD44 confers metastatic potential to rat carcinoma cells
AUTHOR(S): Guenthert, Ursula; Hofmann, Martin; Rudy, Wolfgang; Reber, Sonja; Zoeller, Margot; Haussmann, Irmgard; Matzku, Siegfried; Wenzel, Achim; Ponta, Helmut; Herrlich, Peter
CORPORATE SOURCE: Inst. Genet., Univ. Karlsruhe, Karlsruhe, D-7500, Germany
SOURCE: Cell (Cambridge, MA, United States) (1991), 65(1), 13-24
CODEN: CELLB5; ISSN: 0092-8674
DOCUMENT TYPE: Journal
LANGUAGE: English
AB By using a **monoclonal** antibody (MAb1.1ASML) raised against a surface glycoprotein of the metastasizing rat pancreatic carcinoma cell line BSp73ASML, cDNA **clones** were isolated that encode glycoproteins with partial **homol.** to **CD44**, a presumed adhesion mol. In one of the **clones**, pMeta-1, the epitope marks an addnl. extracellular domain of 162 amino acids inserted into the rat **CD44 protein** between amino acid positions 223 and 247 (by **analogy** to human and murine **CD44**). The new variants are **expressed** only in the metastasizing cell lines of two rat tumors, the pancreatic carcinoma BSp73 and the mammary adenocarcinoma 13762NF; they are not **expressed** in the non-metastasizing tumor cell lines nor in most normal rat tissues. Overexpression of pMeta-1 in the nonmetastasizing BSp73AS cells suffices to establish full metastatic behavior.
IT **136894-51-4**, Antigen **CD 44** (rat **clone** pMeta-1 isoform precursor **protein** moiety reduced)
136894-52-5, Antigen (rat **clone** pMeta-1 isoform **protein** moiety reduced)
RL: PRP (Properties)
(amino acid sequence of)
IT **136896-09-8**, **Deoxyribonucleic acid** (rat **clone** pMeta-1 antigen **CD 44** messenger RNA-complementary)
RL: PRP (Properties)
(nucleotide sequence of)

L29 ANSWER 52 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1990:550260 HCAPLUS
DOCUMENT NUMBER: 113:150260
TITLE: An antibody that facilitates hematopoietic engraftment recognizes CD44
AUTHOR(S): Sandmaier, Brenda M.; Storb, Rainer; Appelbaum, Frederick R.; Gallatin, W. Michael

CORPORATE SOURCE: Div. Clin. Res., Fred Hutchinson Cancer Res. Cent., Seattle, WA, 98104, USA
SOURCE: Blood (1990), 76(3), 630-5
CODEN: BLOOAW; ISSN: 0006-4971
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Pretreatment of recipients with the **monoclonal** antibody (MoAb) S5 facilitates engraftment of bone marrow from mismatched, unrelated donors in the canine transplantation model. In the direct comparisons reported here, the S5 glycoprotein (gp) was found to have structural **homol.** to **CD44** that in humans has been implicated in adhesive interactions of one type of effector cell, the lymphocyte. The S5 antigen and gp90Hermes-1 exhibited codistribution on canine peripheral blood cells. Both S5 and Hermes-1 (anti-**CD44**) MoAbs recognized 90-Kd species in radioimmune pptns. of 125I surface-labeled canine peripheral blood lymphocytes and bone marrow cells. Competitive antibody binding expts. showed that the epitope detected by S5 was distinct from that bound by Hermes-1 but overlapped with those defined by two other known anti-**CD44** reagents, IM7 and Hutch-1. Sequential immunopptn. with S5 and Hermes-1 indicated that the two antibodies recognize the same or overlapping subsets of membrane glycoproteins. Tryptic digestion of S5 and anti-**CD44** immunoppts. generated two major iodinated **peptides** of 27 and 35 Kd in both cases, a further indication of structural homol. Similarly, after N-glycanase digestion, S5 and **CD44** immunoppts. were resolved to a single 68-Kd species. These findings suggest that **CD44**-mediated adhesive events may affect the fat of transplanted hematopoietic cells. The previous implications of this glycoprotein in T-lymphocyte activation and lymphocyte adhesion to endothelium thus provide useful paradigms to analyze its function in the bone marrow transplant setting.

L29 ANSWER 53 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1990:230583 HCAPLUS
DOCUMENT NUMBER: 112:230583
TITLE: The cDNA sequence of mouse Pgp-1 and **homology** to human **CD44** cell surface antigen and proteoglycan core/link **proteins**
AUTHOR(S): Wolffe, E. J.; Gause, W. C.; Pelfrey, C. M.; Holland, S. M.; Steinberg, A. D.; August, J. T.
CORPORATE SOURCE: Sch. Med., Johns Hopkins Univ., Baltimore, MD, 21205, USA
SOURCE: Journal of Biological Chemistry (1990), 265(1), 341-7
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The isolation and sequencing of a cDNA encoding mouse plasma membrane glycoprotein Pgp-1 is reported. An oligonucleotide probe corresponding to the N-terminal sequence of the purified **protein** was synthesized by the polymerase chain reaction and used to screen a mouse macrophage λgt11 library. A cDNA **clone** with an insert of 1.2 kilobases was selected and sequenced. In Northern blot anal., only cells **expressing** Pgp-1 contained mRNA species that hybridized with this Pgp-1 cDNA. The nucleotide sequence of the cDNA has a single open reading frame that yields a **protein**-coding sequence of 1076 base pairs followed by a 133-base pair 3'-untranslated sequence that includes a putative polyadenylation signal but no poly(A) tail. The translated sequence comprises a 13-amino-acid signal **peptide** followed by a **polypeptide** core of 345 residues corresponding to an Mr of 37,000. Portions of the deduced amino acid sequence were identical to those obtained by amino acid sequence anal. from the purified glycoprotein, confirming that the cDNA encodes Pgp-1. The predicted structure of Pgp-1 includes an N-terminal extracellular domain (residues 14-265), a

transmembrane domain (residues 266-286), and a cytoplasmic tail (residues 287-358). Portions of the mouse Pgp-1 sequence are highly similar to that of the human **CD44** cell surface glycoprotein implicated in cell adhesion. The **protein** also shows sequence similarity to the proteoglycan tandem repeat sequences found in cartilage link **protein** and cartilage proteoglycan core **protein** which are thought to be involved in binding to hyaluronic acid.

IT **127384-61-6**, Antigen **CD 44** (mouse **protein** moiety reduced)
RL: PRP (Properties)
(amino acid sequence of)

L29 ANSWER 54 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1990:52016 HCAPLUS
DOCUMENT NUMBER: 112:52016
TITLE: Regulatory signals affecting a selective loss of mRNA in Dictyostelium discoideum
AUTHOR(S): Hassanain, Hamdy H.; Kopachik, Will
CORPORATE SOURCE: Dep. Zool., Michigan State Univ., East Lansing, MI, 48824, USA
SOURCE: Journal of Cell Science (1989), 94(3), 501-9
CODEN: JNCSAI; ISSN: 0021-9533
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Signals that affect mRNA levels complementary to a gene that is highly **expressed** in vegetative D. discoideum cells were identified. This gene has been **cloned** as cDNA in the plasmid pcD-D2. The level of transcripts **homologous** to pcD-D2 **fell** dramatically in strain XP55 during the aggregation stage of development when cells differentiate on agar. The level, however, did not fall simply as a result of starvation or aggregation-specific cell contact. Rather, before the level is reduced cells must be deprived of amino acids and cAMP administered in amts. and at intervals in pulses to mimic cAMP signal-relay in aggregation. This effect can be blocked either with cAMP-S (a nonhydrolyzable cAMP analog) or adenosine, both of which prevent cAMP binding to the cAMP cell surface receptor. It is also blocked in frigid aggregation-deficient mutants HC85 and HC112 known to be defective in a Gα **protein.** Apparently, the transcript level is balanced by pos. nutritional signals acting against neg. signals transduced in part through a cell surface cAMP receptor.

L29 ANSWER 55 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1985:2978 HCAPLUS
DOCUMENT NUMBER: 102:2978
TITLE: Structural and functional analysis of Sendai virus nucleocapsid protein NP with monoclonal antibodies
AUTHOR(S): Deshpande, K. L.; Portner, A.
CORPORATE SOURCE: Dep. Virol. Mol. Biol., St. Jude Child. Res. Hosp., Memphis, TN, 38101, USA
SOURCE: Virology (1984), 139(1), 32-42
CODEN: VIRLAX; ISSN: 0042-6822
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Monoclonal** antibodies specific for Sendai virus nucleocapsid **protein** NP were used to map the antigenic structure of NP and to investigate the role of NP in transcription. Competitive-binding (CB) assays with 9 anti-NP antibodies showed that the NP mol. contained ≥2 topog. distinct sites. By western blot anal., 1 of the NP epitopes that belonged to antigenic site I was localized to a 34,000-mol.-weight (34K) trypsin digest fragment, and another to a (48K) fragment that remained associated with the nucleocapsid. The other antibodies that define antigenic site I did not react with either

fragment; however, the results of CB indicate that their epitopes were in a region on the tertiary structure of the NP mol. that is closely proximal to these fragments. The 48K and 34K fragments have been tentatively identified on the published NP amino acid sequence. Since the 34K and 48K fragments bind antibody, it appears that nucleocapsid-bound NP may be folded into a configuration which places at least some of these sequences on the surface of the nucleocapsid structure. Six antibodies, representing both antigenic sites, were purified for functional studies. All of the antibodies inhibited nucleocapsid transcription in vitro to the same extent (>90%); however, they differed in the amount of antibody required to produce the same effect. Within site I, antibodies producing maximum inhibition were divided into 3 groups: 3 antibodies inhibited at relatively low concns. (0.17 μg); 1 antibody inhibited at an intermediate range (0.43 μg), and another required a 10-fold higher concentration (1.73 μg) to produce the same effect. The antibody which detected the 48K trypsin digest **fragment fell** into the intermediate range for transcription inhibition, whereas the antibody that detected the 34K fragment was in the low range. Thus, antigenic site I, as defined by CB and trypsin digestion studies, can be defined further into 3 subsites which appear to differ in their involvement in the transcription process. Antigenic site II was defined by a single antibody, which also inhibited transcription by >90%.

L29 ANSWER 56 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1982:156622 HCAPLUS
DOCUMENT NUMBER: 96:156622
TITLE: Molecular cloning of vitellogenin gene sequences from Locusta migratoria
AUTHOR(S): Wyatt, G. R.; Locke, J.; Bradfield, J. Y.; White, B. N.; Deeley, R. G.
CORPORATE SOURCE: Dep. Biol., Queen's Univ., Kingston, ON, K7L 3N6, Can.
SOURCE: Developments in Endocrinology (Amsterdam) (1981), 15(Juv. Horm. Biochem.), 299-307
CODEN: DENDD4; ISSN: 0165-1900
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Double-stranded cDNA was prepared from total L. migratoria female fat body RNA, inserted into plasmid pBR322, and **cloned** in Escherichia coli. Screening of the cDNA **clones** by colony hybridization with a probe for female-specific, abundantly **expressed** sequences and establishment of their identity by hybridization with vitellogenin mRNA in Northern blots resulted in the isolation of 4 **clones** that contained vitellogenin sequences. These **clones fell** into 2 **homol.** groups, which probably represented 2 genes. A L. migratoria genomic **DNA** library was prepared by EcoRI digestion of the **DNA**, selection of 10-20-kilobase fragments, in vitro packaging into phage λ Charon 4, and amplification in E. coli using [32P]cDNA prepared from female fat body RNA as a probe, 2 vitellogenin **clones** in the λ phage were isolated. These **clones** appeared to represent nearly identical fragments of the same gene.

L29 ANSWER 57 OF 57 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1971:135155 HCAPLUS
DOCUMENT NUMBER: 74:135155
TITLE: Relation of mRNA of heterogeneous nuclear RNA in mammalian cells
AUTHOR(S): Darnell, James E., Jr.; Pagoulatos, Gerassimos N.; Lindberg, U.; Balint, Robert
CORPORATE SOURCE: Dep. Biol. Sci., Columbia Univ., New York, NY, USA
SOURCE: Cold Spring Harbor Symposia on Quantitative Biology (1970), 35, 555-60
CODEN: CSHSAZ; ISSN: 0091-7451

DOCUMENT TYPE: Journal
LANGUAGE: English
AB When growing cultures of mammalian cells were exposed to radioactive RNA precursors, 2 general types of very high mol. weight nuclear RNA were recognized, ribosomal precursor RNA (r-pre-RNA) and a series of mols., with mol. weight varying between 1.5-8 + 106, which was termed heterogeneous nuclear RNA (HnRNA). The rate of hybridization of the total HnRNA was initially about 5 times that of rRNA. When the HnRNA sequences which hybridized most readily were recovered and tested in a second round of hybridization, they combined with **DNA** at a rate +30 times that of rRNA. HnRNA appeared to consist of a mixture of sequences such that the average ratio was 5 times that of rRNA. Since some of the sequences hybridized 100 times as fast as rRNA, it was suggested that HnRNA consisted of 5% sequences of this type and 95% unique sequences which should hybridize 1/400 as fast as rRNA. Expts. indicated that a spectrum of reiterated sequences from 100 to 0.5 times as reiterated as rRNA were transcribed into HnRNA sequences. The total HnRNA initially hybridized .apprx.3-4 times faster than did the total mRNA. Cells carrying SV40 virus **DNA** integrated in the **host** genome produced Hn-RNA and mRNA, both of which had RNA sequences complementary to SV40 **DNA.** The results **fell** short of proving the **derivation** of mRNA from HnRNA.

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=> d stat que
L1 21 SEA FILE=REGISTRY ABB=ON PLU=ON FASCILIN? OR STABILIN?
L2 379 SEA FILE=REGISTRY ABB=ON PLU=ON CD44 OR CD(L)44
L3 418 SEA FILE=HCAPLUS ABB=ON PLU=ON L1 OR ?FASCILIN? OR ?STABLILIN ?
L4 4329 SEA FILE=HCAPLUS ABB=ON PLU=ON L2 OR CD44 OR CD(W)44
L5 40848 SEA FILE=HCAPLUS ABB=ON PLU=ON FELL
L7 3 SEA FILE=HCAPLUS ABB=ON PLU=ON L3 AND (L4 OR L5)
L10 981 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(L)(?ENCOD? OR CODE? OR CODING OR HOMOLOG?)
L12 42 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(4W)LIKE
L13 6 SEA FILE=HCAPLUS ABB=ON PLU=ON L10 AND L12
L14 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L13 NOT L7
L20 9679 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(L)(EXPRESS? OR ?CLON? OR ?FUSION? OR ?RECOMBIN? OR VECTOR? OR HOST?)
L23 174 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(5A)(?DERIVAT? OR ?ANALOG? OR ?FRAGMENT? OR ?HOMOLOG?)
L24 79 SEA FILE=HCAPLUS ABB=ON PLU=ON L23 AND L20
L25 240 SEA FILE=REGISTRY ABB=ON PLU=ON DEOXYRIBONUCLEIC ACID#/CN OR DNA/CN OR NUCLEIC ACID?/CN
L26 1913 SEA FILE=REGISTRY ABB=ON PLU=ON PROTEIN/CN OR PROTEINS
L27 8456 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(L)(L25 OR ?NUCLEIC(W)ACID OR DNA OR L26 OR PROTEIN OR ?PEPTIDE?)
L28 58 SEA FILE=HCAPLUS ABB=ON PLU=ON L24 AND L27
L29 57 SEA FILE=HCAPLUS ABB=ON PLU=ON L28 NOT (L7 OR L14)
L30 20 SEA FILE=HCAPLUS ABB=ON PLU=ON L24 AND CDNA
L31 1 SEA FILE=HCAPLUS ABB=ON PLU=ON L30 NOT (L7 OR L14 OR L29)
L32 344 SEA FILE=HCAPLUS ABB=ON PLU=ON (L3 OR L4 OR L5)(5A)?SEQUENCE?
L33 30 SEA FILE=HCAPLUS ABB=ON PLU=ON L32 AND L23
L34 7 SEA FILE=HCAPLUS ABB=ON PLU=ON L33 NOT (L7 OR L14 OR L29)
L35 7 SEA FILE=HCAPLUS ABB=ON PLU=ON L31 OR L34

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=>

=> d ibib abs hitrn l35 1-7

L35 ANSWER 1 OF 7 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:179234 HCAPLUS
DOCUMENT NUMBER: 141:34385
TITLE: Construction and analysis of a BAC library in the grass Brachypodium sylvaticum: its use as a tool to bridge the gap between rice and wheat in elucidating gene content
AUTHOR(S): Foote, Tracie N.; Griffiths, Simon; Allouis, Sebastien; Moore, Graham
CORPORATE SOURCE: John Innes Centre, Norwich, NR4 7UH, UK
SOURCE: Functional & Integrative Genomics (2004), 4(1), 26-33
CODEN: FIGUBY; ISSN: 1438-793X
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A BAC library of 30,228 clones with an average insert size of 102 kb was constructed in the grass Brachypodium sylvaticum. Brachypodium has a simple genome, similar in size and repetitive DNA content to that of rice, and is more closely related than rice both to the major temperate cereals wheat and barley, and to the forage grasses. The library represents 6.6 genome equivalent, implying a 99.9% probability of recovering any specific sequence. The library was arrayed onto two high-d. colony filters, which were screened with heterologous DNA probes from rice chromosome nine and from syntenous regions of wheat, barley, maize and oat. The construction of Brachypodium BAC contigs revealed that synteny between rice, wheat and Brachypodium was largely maintained over several regions of rice chromosome nine. This suggests that Brachypodium will be a useful tool in the elucidation of gene content in agronomically important cereal crops, complementing rice as a "grass genome model".
IT **578681-31-9**, GenBank AY343976
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide **sequence**; construction and anal. of BAC library in grass Brachypodium sylvaticum and its use as tool to bridge gap between rice and wheat in elucidating gene content)
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:972193 HCAPLUS
DOCUMENT NUMBER: 140:24172
TITLE: Human cDNA sequences and their encoded proteins and diagnostic and therapeutic uses
INVENTOR(S): Alsobrook, John P., II; Alvarez, Enrique; Anderson, David W.; Boldog, Ferenc L.; Casman, Stacie J.; Catterton, Elina; Chapoval, Andrei; Crabtree-Bokor, Julie R.; Edinger, Shlomit R.
PATENT ASSIGNEE(S): Curagen Corporation, USA
SOURCE: PCT Int. Appl., 1880 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 140
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003102155	A2	20031211	WO 2003-US17430	20030603

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003207394	A1	20031106	US 2002-190115	20020703
US 2004029226	A1	20040212	US 2003-383201	20030306

PRIORITY APPLN. INFO.:

US 2002-385120P	P	20020603
US 2002-385784P	P	20020604
US 2002-386041P	P	20020605
US 2002-386047P	P	20020605
US 2002-386376P	P	20020606
US 2002-386453P	P	20020606
US 2002-386864P	P	20020606
US 2002-387016P	P	20020606
US 2002-386796P	P	20020607
US 2002-386816P	P	20020607
US 2002-386931P	P	20020607
US 2002-386942P	P	20020607
US 2002-386971P	P	20020607
US 2002-387262P	P	20020607
US 2002-296960P	P	20020608
US 2002-387400P	P	20020610
US 2002-387535P	P	20020610
US 2002-387610P	P	20020611
US 2002-387625P	P	20020611
US 2002-387634P	P	20020611
US 2002-387668P	P	20020611
US 2002-387696P	P	20020611
US 2002-387702P	P	20020611
US 2002-387836P	P	20020611
US 2002-387859P	P	20020611
US 2002-387933P	P	20020612
US 2002-387934P	P	20020612
US 2002-387960P	P	20020612
US 2002-388022P	P	20020612
US 2002-388096P	P	20020612
US 2002-389123P	P	20020613
US 2002-389118P	P	20020614
US 2002-389120P	P	20020614
US 2002-389144P	P	20020614
US 2002-389146P	P	20020614
US 2002-389729P	P	20020617
US 2002-389742P	P	20020617
US 2000-215854P	P	20000703
US 2000-215856P	P	20000703
US 2000-215902P	P	20000703
US 2000-216585P	P	20000707
US 2000-216586P	P	20000707
US 2000-216722P	P	20000707
US 2000-218622P	P	20000717
US 2000-218992P	P	20000717
US 2000-221285P	P	20000727
US 2001-268734P	P	20010214
US 2001-274260P	P	20010308
US 2001-279856P	P	20010329
US 2001-898994	A1	20010703

US 2001-303168P	P	20010705
US 2002-51874	A	20020116
US 2002-361974P	P	20020306
US 2002-93463	A	20020308
US 2002-365034P	P	20020315
US 2002-365477P	P	20020319
US 2002-365884P	P	20020320
US 2002-365984P	P	20020320
US 2002-365985P	P	20020320
US 2002-366928P	P	20020322
US 2002-368996P	P	20020401
US 2002-372018P	P	20020412
US 2002-372022P	P	20020412
US 2002-374682P	P	20020423
US 2002-389143P	P	20020614
US 2002-391779P	P	20020626
US 2002-403743P	P	20020815
US 2002-410755P	P	20020913
US 2002-412957P	P	20020923
US 2002-420382P	P	20021022

AB Disclosed herein are 62 cDNA sequences that encode novel human polypeptides that are members of various protein families. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivs., variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

L35 ANSWER 3 OF 7 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:964486 HCAPLUS
DOCUMENT NUMBER: 138:34137
TITLE: A knockout mouse with the endogenous α4 integrin gene inactivated and compensated by a chemically regulated α4 integrin gene and its use in assays for α4 integrin antagonists and modulators of VLA4 signaling
INVENTOR(S): Wasel-Nielen, Monika; Kirschbaum, Bernhard; Foster, Martyn; Polites, Gregory; Khorkova, Olga; Zhu, Bin
PATENT ASSIGNEE(S): Aventis Pharmaceuticals Inc., USA
SOURCE: PCT Int. Appl., 175 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002101017	A2	20021219	WO 2002-US18477	20020607
WO 2002101017	A3	20030904		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003154499 A1 20030814 US 2002-163899 20020605
EP 1406997 A2 20040414 EP 2002-741981 20020607
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRIORITY APPLN. INFO.: US 2001-297112P P 20010608
GB 2001-24895 A 20011017
US 2002-382927P P 20020523
US 2002-384109P P 20020529
US 2002-163899 A 20020605
WO 2002-US18477 W 20020607

AB Provided herein is a mouse that is unable to express functional alpha-4-integrin protein, and methods for assaying agents for alpha-4 integrin antagonist activity, as well as genetic markers for analyzing the efficacy of VLA-4 modulators, and particularly antagonists. An internally compensated homozygous α4 integrin knockout mouse that develops normally in the womb and that can be used in assays for modulators and effectors of α4 integrin function are described. The endogenous α4 integrin genes are knocked out and compensated for by a replacement gene that is under control of a tetracycline-regulated promoter. By exposing the pregnant female to tetracycline, α4 integrin gene expression is assured during development, allowing normal development of the fetus. Mice no longer expressing the α4 integrin gene showed abnormalities in the development of hematopoietic cells and of the bone marrow and venous system. Anal. of patterns of gene expression in knockout and control littermates showed up- and down-regulation of gene expression.

L35 ANSWER 4 OF 7 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:429846 HCAPLUS
DOCUMENT NUMBER: 135:164882
TITLE: Mouse LYVE-1 is an endocytic receptor for hyaluronan in lymphatic endothelium
AUTHOR(S): Prevo, Remko; Banerji, Suneale; Ferguson, David J. P.; Clasper, Steven; Jackson, David G.
CORPORATE SOURCE: Medical Research Council Human Immunology Unit, Institute of Molecular Medicine, John Radcliffe Hospital, Oxford, OX3 9DS, UK
SOURCE: Journal of Biological Chemistry (2001), 276(22), 19420-19430
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The glycosaminoglycan hyaluronan is a key substrate for cell migration in tissues during inflammation, would healing, and neoplasia. Unlike other matrix components, hyaluronan (HA) is turned over rapidly, yet most degradation occurs not locally but within distant lymph nodes, through mechanisms that are not yet understood. While it is not clear which receptors are involved in binding and uptake of hyaluronan within the lymphatics, one likely candidate is the lymphatic endothelial hyaluronan receptor LYVE-1 recently described in our laboratory Here we present evidence that LYVE-1 is involved in the uptake of hyaluronan by lymphatic endothelial cells using a new murine LYVE-1 orthologue identified from the EST data base. We show that mouse LYVE-1 both binds and internalizes hyaluronan in transfected 293T fibroblasts in vitro and demonstrate using immunoelectron microscopy that it is distributed equally among the luminal and abluminal surfaces of lymphatic vessels in vivo. In addition, we show by means of specific antisera that **expression** of mouse LYVE-1 remains restricted to the lymphatics in homozygous knockout mice lacking a functional gene for **CD44**, the closest **homolog** of LYVE-1 and the only other Link superfamily HA receptor known to date.

Together these results suggest a role for LYVE-1 in the transport of HA from tissue to lymph and imply that further novel hyaluronan receptors must exist that can compensate for the loss of **CD44** function.
REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:69856 HCAPLUS
DOCUMENT NUMBER: 130:148692
TITLE: Anti-inflammatory and antimetastatic CD44 peptides that inhibit T-cell activation
INVENTOR(S): Haynes, Barton F.; Patton, Karen L.; Liao, Hua-Xin
PATENT ASSIGNEE(S): Duke University, USA
SOURCE: U.S., 35 pp., Cont.-in-part of U.S. Ser. No. 973,339, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5863540	A	19990126	US 1993-143311	19931029
US 6432405	B1	20020813	US 1996-753851	19961202

PRIORITY APPLN. INFO.:
US 1991-669730 B2 19910315
US 1992-973339 B2 19921030
US 1991-682518 B1 19910409
US 1992-945581 B1 19920916
US 1993-47068 B1 19930416

AB The present invention relates, in general, to a method of treating inflammation or inhibiting cancer cell metastasis. In particular, the present invention relates to a method of suppressing T cell activation, inhibiting CD44-mediated cell adhesion and CD44-monocyte IL1 release, treating inflammation, and transporting a drug to a site of inflammation.
IT **157147-26-7 157147-27-8 157147-28-9**
157147-30-3 157147-32-5 157147-33-6
157147-34-7 157147-36-9 157153-40-7
157172-83-3 157242-80-3 157242-82-5
157242-88-1
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PEP (Physical, engineering or chemical process); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(amino acid **sequence**; anti-inflammatory and antimetastatic **CD44** peptides that inhibit T-cell activation)
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:39093 HCAPLUS
DOCUMENT NUMBER: 128:127034
TITLE: Identification of CD44 residues important for hyaluronan binding and delineation of the binding site
AUTHOR(S): Bajorath, Jurgen; Greenfield, Brad; Munro, Sandra B.; Day, Anthony J.; Aruffo, Alejandro
CORPORATE SOURCE: Bristol-Myers Squibb Pharmaceutical Research Institute, Seattle, WA, 9812, USA
SOURCE: Journal of Biological Chemistry (1998), 273(1), 338-343
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular

Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB CD44 is a widely distributed cell surface protein that plays a role in cell adhesion and migration. As a proteoglycan, CD44 is also implicated in growth factor and chemokine binding and presentation. The extracellular region of CD44 is variably spliced, giving rise to multiple CD44 isoforms. All isoforms contain an amino-terminal domain, which is homologous to cartilage link proteins. The cartilage link protein-like domain of CD44 is important for hyaluronan binding. The structure of the link protein domain of TSG-6 has been determined by NMR. Based on this structure, a mol. model of the link-**homologous** region of **CD44** was constructed. This model was used to select residues for site-specific mutagenesis in an effort to identify residues important for ligand binding and to outline the hyaluronan binding site. Twenty-four point mutants were generated and characterized, and eight residues were identified as critical for binding or to support the interaction. In the model, these residues form a coherent surface the location of which approx. corresponds to the carbohydrate binding sites in two functionally unrelated calcium-dependent lectins, mannose-binding protein and E-selectin (CD62E).
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:470119 HCAPLUS
DOCUMENT NUMBER: 127:92423
TITLE: Method of diagnosing and treating carcinoma
INVENTOR(S): Heider, Karl-Heinz; Adolf, Gunther; Ostermann, Elinborg; Patzelt, Erik; Sproll, Marlies
PATENT ASSIGNEE(S): Boehringer Ingelheim International Gmbh, Germany; Forschungszentrum Karlsruhe Gmbh; Heider, Karl-Heinz; Adolf, Gunther; Ostermann, Elinborg; Patzelt, Erik; Sproll, Marlies
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9721104	A1	19970612	WO 1996-EP5448	19961205
W: AU, BG, BR, BY, CA, CN, CZ, EE, HU, IL, JP, KR, KZ, LT, LV, MX, NO, NZ, PL, RO, RU, SG, SK, TR, UA, US, UZ, VN				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
DE 19545472	A1	19970612	DE 1995-19545472	19951206
AU 9711773	A1	19970627	AU 1997-11773	19961205
AU 726704	B2	20001116		
EP 865609	A1	19980923	EP 1996-942362	19961205
EP 865609	B1	20030319		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, LT, LV, FI				
BR 9611901	A	19990302	BR 1996-11901	19961205
JP 2000502067	T2	20000222	JP 1997-520993	19961205
NZ 324314	A	20000228	NZ 1996-324314	19961205
EE 3783	B1	20020617	EE 1998-164	19961205
RU 2193779	C2	20021127	RU 1998-112600	19961205
PL 184521	B1	20021129	PL 1996-327066	19961205
AT 235056	E	20030415	AT 1996-942362	19961205
NO 9802588	A	19980805	NO 1998-2588	19980605

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     BG 62985          B1   20001229       BG 1998-102513   19980605
     HK 1011560        A1   20031121       HK 1998-112910   19981207
PRIORITY APPLN. INFO.:                   DE 1995-19545472 A  19951206
                                         DE 1996-19615074 A  19960417
                                         WO 1996-EP5448   W  19961205
AB   The invention concerns a method of diagnosing and treating carcinomas, the
     method being based on the expression of the variant exon v6 of the CD44
     gene as the mol. target.  In a preferred embodiment, v6-specific antibody
     mols., in particular the monoclonal antibody BIWA-1 (VFF-18), are used for
     this purpose.
IT   161309-27-9
     RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL
     (Biological study); USES (Uses)
        (nucleotide sequence; CD44 gene exon v6 in
        carcinoma diagnosis and therapy)


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E13 THROUGH E72 ASSIGNED

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E73 THROUGH E87 ASSIGNED

=> fil hcaplus
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FILE COVERS 1907 - 11 Jul 2004  VOL 141 ISS 3
FILE LAST UPDATED: 9 Jul 2004  (20040709/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


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L38 70 S L37 AND (L1 OR L2)

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6 RN 500377-29-7 REGISTRY
7 RN 500377-28-6 REGISTRY
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9 RN 500377-26-4 REGISTRY
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36 RN 157242-80-3 REGISTRY
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38 RN 157153-40-7 REGISTRY
39 RN 157147-44-9 REGISTRY
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70        RN   127384-61-6  REGISTRY


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L38  ANSWER 1 OF 70  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   578681-31-9  REGISTRY
CN   DNA (Brachypodium sylvaticum fascilin gene sequence homolog fragment)
     (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   GenBank AY343976
FS   NUCLEIC ACID SEQUENCE
MF   Unspecified
CI   MAN
SR   GenBank
LC   STN Files:   CA, CAPLUS, GENBANK
DT.CA  CAplus document type:  Journal
RL.NP  Roles from non-patents:  BIOL (Biological study); PRP (Properties)

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               1 REFERENCES IN FILE CA (1907 TO DATE)
               1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE   1:  141:34385

L38  ANSWER 10 OF 70  REGISTRY  COPYRIGHT 2004 ACS on STN
RN   500377-25-3  REGISTRY
CN   DNA (chicken clone WO-03/018044-SEQID5 CD44-hyaluronic acid-binding
     domain fragment-specifying cDNA) (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   5: PN: WO03018044 SEQID: 5 claimed DNA
FS   NUCLEIC ACID SEQUENCE
MF   Unspecified
CI   MAN
SR   CA
LC   STN Files:   CA, CAPLUS, TOXCENTER
DT.CA  CAplus document type:  Patent
RL.P   Roles from patents:  BIOL (Biological study); PREP (Preparation); PRP
       (Properties); USES (Uses)

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1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 138:198601

L38 ANSWER 20 OF 70 REGISTRY COPYRIGHT 2004 ACS on STN
RN **224340-17-4** REGISTRY
CN INDEX NAME NOT YET ASSIGNED
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: CA, CAPLUS, GENBANK
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PRP (Properties)

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1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 139:291131

L38 ANSWER 30 OF 70 REGISTRY COPYRIGHT 2004 ACS on STN
RN **203673-44-3** REGISTRY
CN **DNA (human clone HUVDE75 CD44 (antigen) cDNA plus flanks) (9CI)** (CA INDEX NAME)
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); OCCU (Occurrence); PRP (Properties); USES (Uses)

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2 REFERENCES IN FILE CAPLUS (1907 TO DATE)

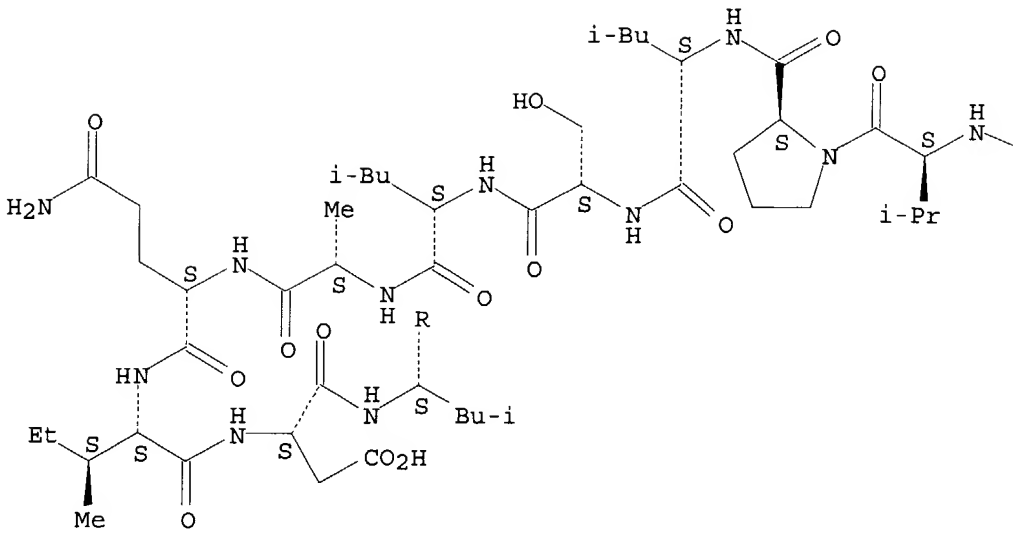
REFERENCE 1: 131:154496

REFERENCE 2: 128:189203

L38 ANSWER 40 OF 70 REGISTRY COPYRIGHT 2004 ACS on STN
RN **157147-43-8** REGISTRY
CN L-Asparagine, L-methionyl-L-α-aspartyl-L-lysyl-L-phenylalanyl-L-tryptophyl-L-tryptophyl-L-histidyl-L-alanyl-L-alanyl-L-tryptophylglycyl-L-leucyl-L-cysteinyl-L-leucyl-L-valyl-L-prolyl-L-leucyl-L-seryl-L-leucyl-L-alanyl-L-glutaminyl-L-isoleucyl-L-α-aspartyl-L-leucyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN **CD44 Antigen fragment (human)**
FS PROTEIN SEQUENCE; STEREOSEARCH
MF C139 H203 N33 O33 S2
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study)

Absolute stereochemistry.

PAGE 1-A



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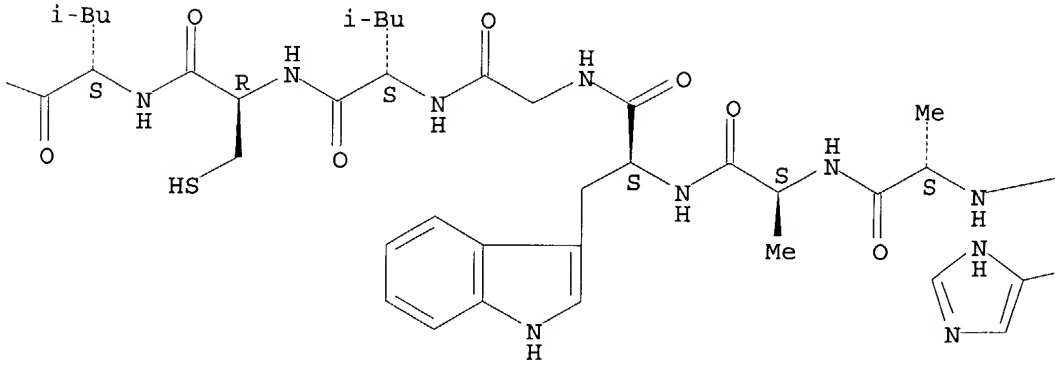

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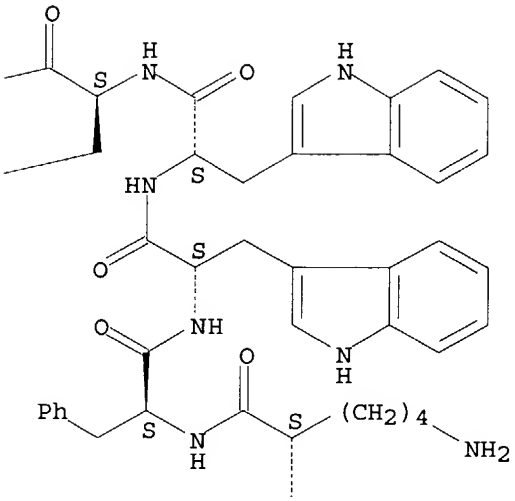

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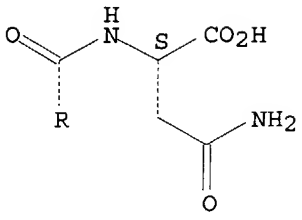

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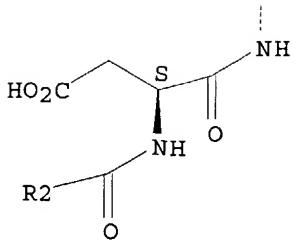

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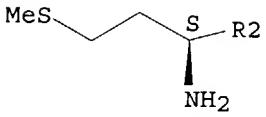


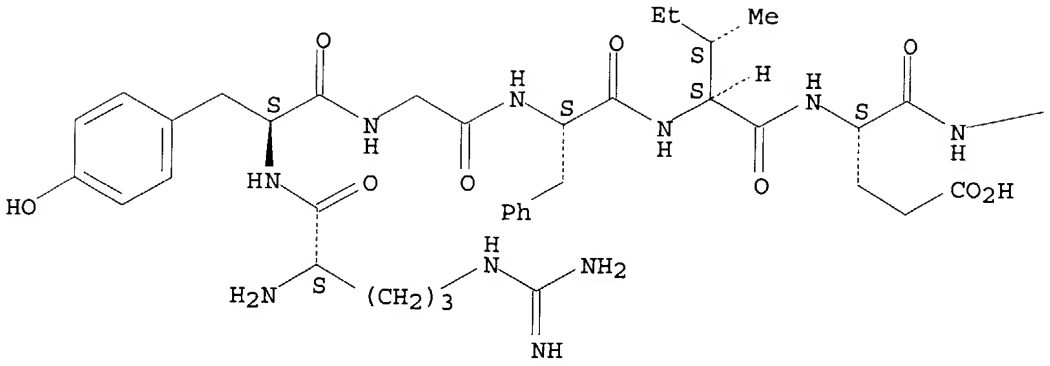
1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 121:155742

L38 ANSWER 50 OF 70 REGISTRY COPYRIGHT 2004 ACS on STN
RN **157147-33-6** REGISTRY
CN L-Isoleucine, L-arginyl-L-tyrosylglycyl-L-phenylalanyl-L-isoleucyl-L-α-glutamylglycyl-L-histidyl-L-valyl-L-valyl-L-isoleucyl-L-prolyl-L-arginyl-L-isoleucyl-L-histidyl-L-prolyl-L-asparaginyl-L-seryl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1060: PN: WO02078524 SEQID: 1295 unclaimed protein
CN **CD44 Antigen fragment (human)**
FS PROTEIN SEQUENCE; STEREOSEARCH
MF C102 H158 N30 O25
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: BIOL (Biological study); PROC (Process); PRP (Properties); USES (Uses)

Absolute stereochemistry.

PAGE 1-A



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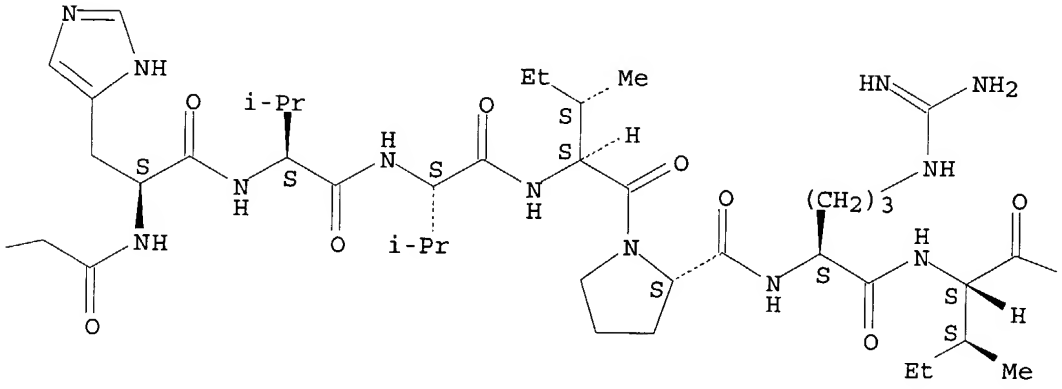

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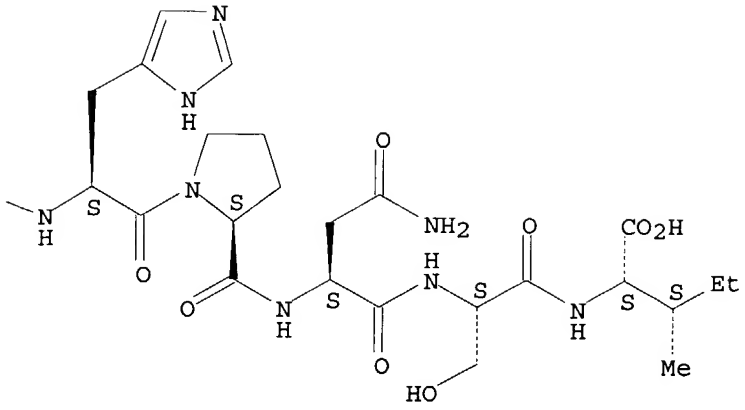

3 REFERENCES IN FILE CA (1907 TO DATE)
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REFERENCE 1: 137:274808

REFERENCE 2: 130:148692

REFERENCE 3: 121:155742

L38 ANSWER 60 OF 70 REGISTRY COPYRIGHT 2004 ACS on STN
RN **148790-22-1** REGISTRY
CN **DNA (rat BSpASML cell antigen CD 44-specifying fragment) (9CI)** (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN **Deoxyribonucleic acid (rat BSpASML cell antigen CD 44-specifying fragment)**
OTHER NAMES:
CN **DNA (rat tumor cell line BSpASML CD44 antigen variant cDNA)**
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL
DT.CA CAplus document type: Patent
RL.P Roles from patents: PRP (Properties)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
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1 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 119:70365

L38 ANSWER 70 OF 70 REGISTRY COPYRIGHT 2004 ACS on STN
RN **127384-61-6** REGISTRY
CN **Antigen CD 44 (mouse protein moiety reduced) (9CI)** (CA INDEX NAME)
FS PROTEIN SEQUENCE
MF Unspecified
CI MAN
SR CA

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LC   STN Files:   CA, CAPLUS, USPATFULL
DT.CA  CAplus document type:  Journal
RL.NP  Roles from non-patents:  PRP (Properties)

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               1 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE   1:  112:230583


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